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HANDBOOK

OF

PRACTICAL MEDICINE

BY

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VOLUME IV.

DISEASES OF THE BLOOD AND NUTRITION, AND INFECTIOUS DISEASES

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HANDBOOK

OF

PRACTICAL MEDICINE.

SECTION VIII.

DISEASES OF THE BLOOD AND THE HEMATOPOIETIC ORGANS.

PART I.

DISEASES OF THE BLOOD.

1. *Leukæmia. Leucocythæmia.*

I. Etiology.—Leukæmia consists of a constantly increasing excess of white blood-globules in the blood, with progressive diminution in the number of red blood-globules.

We distinguish splenic, lymphatic, and myelogenic leukæmia, according as the spleen, lymphatic glands, or medulla of the bones form the starting-point of the disease. As a rule, we have to deal with mixed forms of the disease, although the organs concerned in the production of blood are often affected in very different degrees.

Béhier also recognizes enteric leukæmia, in which the follicular apparatus of the intestines is said to form the starting-point of the disease. In Béhier's case the spleen and lymphatic glands were unchanged, while extensive hyperplastic changes were visible in the lymph gland apparatus of the intestines, but no examination of the medulla of the bones was made, so that the case was possibly myelogenic in its origin.

Neumann has shown that in the majority, perhaps in all, of the cases the medulla of the bones is the primary starting-point, but the changes are rarely limited to this part. Splenic lymphatic changes supervene in the majority of cases.

Leukæmia is more frequent in men (among 200 cases, 135 males, 65 females).

The disease occurs usually between the twentieth and fiftieth years, the maximum occurring in males in the third decennium, in females in the fourth decennium. It is also observed in children and old people (oldest case at seventy-three years). In childhood it is more frequent between the seventh and fourteenth years.

There is no doubt that the laboring classes are attacked with special frequency.

In not a few cases no exciting cause is demonstrable. The disease sometimes follows injuries to the spleen and bones. Mursick reported a case of acute development of leukæmia five days after amputation of the thigh.

The disease has also been attributed occasionally to bodily and mental strain, grief, care, and excitement, and alcoholic excesses.

Pregnancy, delivery, and menstrual disturbances are supposed to be etiologically related to leukæmia, and Paterson states that in such cases the morbid changes may run a very acute course.

The disease has also been observed after chronic diarrhœa, and in rachitic and scrofulous children.

It may also follow infectious diseases. Chief among these is malaria, particularly irregular, chronic cases. Syphilis sometimes acts as a cause, especially hereditary syphilis in children. Immermann observed a case of myelogenic leukæmia after typhoid fever. Diphtheria is also said to be an occasional etiological factor. Whether progressive pernicious anæmia should be included among the causes is still doubtful. Two cases have been reported in which this disease was converted into leukæmia.

Hereditary influences seem to have been potent in a few cases.

Casati observed splenic leukæmia in a girl whose grandmother and father had suffered from the same disease. Biermer observed it in two sisters, æt. three years and four and one-half years, Senator in twins, æt. one and one-half years.

The disease occurs in animals (dog, horse, cow, pig).

II. Symptoms.—The most striking symptoms are the changes in the blood. All other symptoms may occur under other circumstances, particularly in anæmic conditions. To examine the blood, the tip of the finger, after having been cleaned, is pricked with a needle, a small drop of blood received upon a clean cover glass, and this placed upon an object glass. If the surfaces of the glass are clean, the blood will be distributed in a uniform laver.

The blood often presents macroscopic peculiarities. It is unusually light and watery, sometimes chocolate-brown or yeast colored, and coagulates slowly. If a larger amount is removed by cupping, white streaks and dots are noticed on the clot, or it is covered with a whitish gray layer, which consists of white blood-globules. Under the microscope it is found that, instead of the normal proportion of white blood-globules (1 to 350–500 red blood-globules), they are increased to such an extent that they sometimes equal, or even exceed, the red blood-globules. In a few very advanced cases, a certain amount of care was requisite in order to discover any red blood-globules. Even in less advanced cases, the microscopic appearances are very charactêristic (vide Fig. 1).

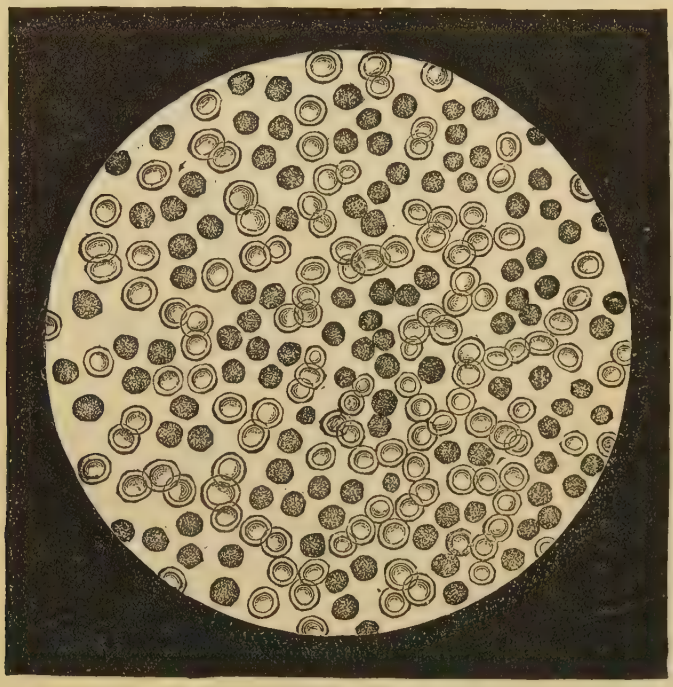
Three principal forms of white blood-globules are visible. One form is smaller than the red blood-globules and contains a single nucleus surrounded by a narrow zone of protoplasm. The latter is sometimes so small in amount that the cell looks like a free nucleus. These white

blood-globules resemble the parenchyma cells of the lymphatic glands, and abound in leukæmia of a predominantly lymphatic character.

A second form of white blood-globules exceeds the red ones in size. They contain generally three or four nuclei, sometimes are grouped together like a clover leaf, and often present constrictions. This form resembles the cells of the splenic pulp, are especially numerous in leukæmia of a predominantly splenic character, and are probably derived in great part from the spleen.

Finally, Mosler called attention to white blood-globules which contain drops of fat. They are said to be derived from the medulla of the bones, and to be characteristic of myelogenic leukæmia. Much more

FIG. 1.



Blood in leukæmia (chiefly lymphatic). Enlarged 450 times.

characteristic of the latter form, however, are the so-called transition cells of E. Neumann. These are imperfectly developed red blood-globules which still contain a large nucleus, while the zonal portion is homogeneous and colored.

Various forms of white blood-globules are found even in the blood of healthy individuals. This should be expected *à priori* in leukæmia, because, as a general thing, we do not observe pure forms of lymphatic, splenic, or myelogenic leukæmia. Recent investigations have shown, however, that the form of the white blood-globules is not a positive indication of their place of origin, and that a still larger variety of these cells may be distinguished by staining with aniline.

The red blood-globules are sometimes very much diminished in num-

ber, and they may even be reduced to half a million in one cubic millimetre (normally five millions). Their number may vary greatly in different examinations. They may be pale and abnormal in shape (pear shaped, club shaped, etc.), so-called poikilocytosis.

The frequent spontaneous hemorrhages of leukæmia furnish abundant material for examination. The blood is not infrequently cloudy, milky, or pus-like. It readily decomposes and becomes acid, probably as the result of the formation of glycerin-phosphoric acid from the lecithin contained in it. The specific gravity is diminished to 1.036 to 1.049 (normally 1.055). It possesses slight tendency to coagulation. According to Bockendahl and Landwehr, this is owing to the large proportion of peptone which is said to be derived from the white blood-globules. If the blood is allowed to stand in a test-tube, the lowest layer of sediment, consisting of red globules, is diminished, the middle layer of white globules is unusually large. The longer the blood is allowed to stand the more abundant the crystals which are deposited (Charcot-Neumann crystals). Zenker attributes their origin to the white blood-globules in and upon which he has found them (vide Fig. 2).

The amœboid movements of the white blood-globules are sometimes diminished or lost. According to Birk, they produce no fibrin ferment. Jæderholm reports a case in which many of them were filled with fine fat granules, so that the nucleus was often concealed. If the disease has been preceded by intermittent fever, they sometimes contain pigment granules (melanoleukæmia). In one case, Friedreich observed amœboid movements of the red blood-globules. In some cases, there is a large number of protoplasm granules (hæmatoblasts), often aggregated in groups. Unusually small red blood-globules (microcytes) have been repeatedly seen. A few writers claim that schizomycetes are present in the blood.

Fig. 2.

Leukæmic crystals from the blood, partly free, partly inclosed in white blood-globules. After Zenker.

Quincke found only one-third of the normal amount of hæmoglobin in the blood, but, according to Laacher, the quantity in the individual red blood-globules is unchanged.

Leukæmic blood contains substances which appear to be derived, in great part, from the blood-producing organs. We may mention hypoxanthin, xanthin, glutin, lecithin, formic acid, lactic acid, succinic acid, peptone, traces of leucin, and a phosphorus-containing organic acid probably glycerin-phosphoric acid). Hypoxanthin and glutin are characteristic of leukæmia. The former has been found in healthy blood, but only after standing for some time. It is probably derived in great part from the spleen, but may also be found in lymphatic leukæmia. Neumann and Salkowski are inclined to attribute the presence of glutin to changes in the medulla of the bones.

Next in clinical importance to the abnormalities of the blood are the local changes in the blood-producing organs.

Changes in the spleen are the most constant. The organ is usually very much enlarged, and is sometimes tender on pressure. Peritonitic friction murmurs are sometimes felt on palpation of the spleen, and auscultation reveals vascular murmurs, coincident with the pulse, and similar in character to uterine murmurs. The spleen may be so large as to displace adjacent organs, and to give rise to rupture and death from perforation-peritonitis. The organ constantly increases in size, though temporary diminution sometimes occurs after obstinate diarrhœa and profuse hemorrhages.

The enlarged lymphatic glands may attain the size of a fist or even more, and often protrude under the skin as flat prominences. In the neck, they give rise to great deformity and interfere with the mobility of the head and neck. Large glands are often observed in the axillæ and inguinal folds. The cervical and submaxillary glands are

first attacked. As a rule, the tumors are not tender on pressure, and, unlike scrofulous glands, the overlying skin is not reddened or adherent; furthermore, the swelling is generally flatter and softer. As the disease advances, the consistence of the tumors not infrequently increases. Caseation and suppuration are extremely rare in leukæmic lymphatic glands.

The spleen and glands are often enlarged long before the blood undergoes leukæmic changes.

The internal lymphatic glands often undergo hyperplasia. Swelling of the tracheal and bronchial glands is sometimes shown by slight prominence of the manubrium sterni and dulness on percussion, or by the signs of tracheal or bronchial stenosis, as the result of compression (inspiratory retraction of the intercostal spaces, stenotic murmurs, cyanosis, objective and subjective dyspnœa). Pressure upon the œsophagus may interfere with deglutition. Some authors have attributed attacks of palpitation to compression of the pneumogastric. Compression of the recurrent laryngeal is followed by paralysis of the vocal cords. On pressing the hand deep in the abdomen, the mesenteric and retroperitoneal glands may be felt as nodular tumors.

Enlargement of the tonsils, thyroid gland, and even of the persistent thymus gland is observed in some patients. Hyperplasia of the lymph follicles at the base of the tongue has also been noticed.

Affection of the medulla of the bones is sometimes, though not constantly, manifested by pain over the bones, particularly the sternum and spine, and occasionally the long bones. We sometimes find slight depressions in the bones, and soft, yielding places.

Less important symptoms are presented by the urine. Its amount is generally normal, though it is sometimes increased or diminished. It is often pale and has an acid reaction. The specific gravity is generally unchanged. It sometimes contains shining crystals of pure uric acid, and deposits a uric-acid sediment. The amount of urea may be increased or diminished; its amount probably increases with the degree of cachexia. The amount of uric acid is increased. In healthy individuals the proportion of uric acid to urea is 1 : 50–80; in leukæmia, Salkowski found it 1 : 16. According to Fleischer and Penzoldt, the excretion of phosphoric and sulphuric acids is increased, that of lime unchanged. Salkowski discovered traces of formic acid, and diminution of oxalic acid. The urine sometimes contains small amounts of albumin, and occasionally casts. According to some authors, an abundant sediment of round cells indicates lymphomatous deposits in the kidneys (?).

R. Liebreich showed that the retina presents characteristic changes (retinitis leucæmica) in one-fourth or one-third of the cases.

The retina is often pale, and has an orange-yellow color. The veins are wide, sinuous, and rosy red; in places they are bordered with white. The retinal arteries are narrow and pale yellow. The retina is sometimes cloudy; the boundaries of the papilla may be indistinct, especially on the nasal side. There are more or less numerous retinal hemorrhages. Special attention is merited by prominent yellow patches, which are not infrequently surrounded by a red ring of extravasation of blood. Leber states that they are found particularly in the peripheral portions of the retina between the equator and ora serrata, next in the vicinity of the macula lutea. Visual disturbances may be entirely absent, but if the macula lutea is affected, the interference with vision may attract attention before the other symptoms.

Rarer phenomena are hemorrhages into the vitreous, or hemorrhages and lymphomatous formations in the choroid and iris. Leber has described exophthalmus and large lymphomatous formations in the eyelids. Birk has observed bilateral exophthalmus as the result of lymphomata in the posterior portions of the orbits. Similar growths may develop in the lachrymal glands. The development of cataract is sometimes attributed to leukæmia.

Auditory disturbances have been observed in leukæmia, and in one case Pulitzer found lymphomatous deposits in both labyrinths.

A rare but characteristic sign is the appearance of leukæmic tumors in the skin and epididymis.

A series of other symptoms may develop which are the result of anæmia, rather than of the leukæmia.

The first symptoms generally consist of increasing pallor and feebleness. Other patients complain, at an early period, of stitches in the splenic region and of occasional attacks of fever.

When the patients come under our observation, they generally attract attention by the striking pallor of the skin and mucous membranes. The complexion is often dirty gray; in a few cases, the skin is jaundiced. The panniculus adiposus is sometimes very well developed, but in the later stages it undergoes emaciation. There is often an unusual tendency to sweating, occasionally hectic night sweats. Furunculosis or the development of bullous and pustular eruptions has also been described. Cutaneous œdema is observed not infrequently, at first temporarily, later permanently.

The bodily temperature is not infrequently elevated, the type of fever being irregular. The pulse is usually soft and accelerated. Many patients complain of dyspnœa, which is often noticeable objectively. This is the result of the diminution in the number of red blood-globules, the interference of the splenic tumor with the movements of the diaphragm and thorax, feebleness of the heart, and sometimes of compression of the trachea or bronchi by the enlarged thyroid, thymus, or lymphatic glands.

The sensorium may remain entirely intact. In some patients, however, delirium sets in, terminating in mania, and must be regarded as the result of defective nutrition of the brain.

There is a tendency to catarrh of the air passages, and pneumonia is not an infrequent fatal complication. In later stages, serous fluid may accumulate in the pleural cavities.

Pettenkofer and Voit found that, despite the diminished number of red blood-globules, the absorption of oxygen and excretion of carbonic acid remained normal. The excretion of water and urea was greater at night than during the day, unlike what obtains in healthy individuals.

Anæmic systolic murmurs are heard not infrequently over the heart. The organ is often dilated, particularly the right half, and is pushed upwards by the enlarged spleen. Attacks of palpitation sometimes occur spontaneously or after slight causes. The jugular vein may be distended occasionally on one side alone, as the result of compression by enlarged glands. The venous pulse is often present.

Appetite is generally lost, while thirst is often increased. The parotid and submaxillary glands are sometimes increased in size, as the result of lymphomatous deposits. Leukæmic stomatitis and pharyngitis may set in and render deglutition painful. A feeling of pressure in the

stomach, eructations, and vomiting are not infrequent. A more serious symptom is obstinate diarrhœa; this proves fatal in some cases. Virchow found a large amount of leucin and tyrosin in the passages. The liver is almost always enlarged, as the result of lymphomatous infiltrations. Ascites develops occasionally.

Priapism has been observed in a number of cases.

A noteworthy symptom is the tendency to hemorrhages. These may take place beneath the skin, into the muscles, or from the mouth, nose, air passages, gastro-intestinal tract, and genito-urinary apparatus. Kuestner described a case in which sudden hemorrhage into the abdominal muscles produced symptoms of peritonitis, and terminated fatally. Hemorrhages into the brain produce the ordinary symptoms of cerebral apoplexy. Pepper observed sudden deafness as the result of hemorrhage into the ear.

In one case, Eisenlohr described bulbar symptoms. May reported peripheral facial paralysis as the result of lymphomatous infiltration of the sheath of the nerve.

The disease is generally chronic, and may even last eight years.

The average duration is one to two years. Acute cases have also been reported. In one case death occurred upon the eighteenth day of the disease; in another, upon the twenty-fifth day.

Steinberg and Schultze noticed a cadaverous odor several hours before death, and the rapid occurrence of emphysema of the skin and internal organs soon after death.

Death may be the result of increasing marasmus, or of unforeseen complications (pneumonia, hemorrhage, rupture of the suprarenal capsules, cerebral hemorrhage, etc.). Friedlaender reported a case in which the signs of cerebral tumor appeared, as the result of lymphomatous new-formations in the brain.

III. Anatomical Changes.—Lymphomatous new-formations in leukæmia occur either as true hyperplasiæ in localities which contain lymph follicles, or they develop independently of them in a heteroplastic manner. They appear to have a double mode of development. In part they are the result of abnormally profuse diapedesis and extravasation of white blood-globules from the vessels, in part of proliferation of pre-existing connective-tissue cells. They appear as a diffuse infiltration or as nodular formations. The latter may be so small as to resemble tubercles, but they are hardly ever cheesy and do not contain tubercle bacilli.

The peripheral lymphatic glands often form large tumors, which are white or speckled grayish red on section. In recent cases, they are soft and succulent; in older ones, they are harder. They depend on hyperplasia of the cellular elements, particularly of the cortical substance; this is followed by increase of the interstitial connective tissue and increased consistence of the tumor.

The pericardial cavity generally contains serous transudation, which is occasionally sanguinolent, as the result of hemorrhages.

Nodular or more diffuse lymphomata may be found beneath the epicardium, generally in the immediate vicinity of the vessels.

The left heart is usually empty. The blood contained in the right heart sometimes looks exactly like pus; there is an unusually small amount of blood in the venæ cavæ, and in all the other organs.

As a rule, the heart muscle is pale, occasionally infiltrated with small hemorrhages and fatty in places. It may also contain lymphomatous new-formations.

The pleural cavities generally contain transudation. In two cases, I found lymphomatous growths in the pleura, in one case in a diffuse, in the other in a nodular form. Similar growths may occur on the epiglottis, beneath the mucous membrane of the larynx, trachea, and bronchi, in the pulmonary interstitial tissue and alveoli. Boetteher described degeneration of the lymphomata and rupture into the bronchi, giving rise to the formation of a cavity. The tracheal and bronchial glands are converted not infrequently into tumors as large as a fist. The thyroid and thymus glands may also be very much enlarged and infiltrated with lymphomata.

Ascitic fluid is often found in the abdomen, and the peritoneum is sometimes strewn with nodular or diffuse lymphomata.

The spleen often occupies the larger part of the abdominal cavity. In Sixer's case it weighed 16½ pounds, and was 37 cm. long, 25 cm. broad. It is often adherent to adjacent organs. The capsule is generally thickened, as hard as cartilage in places, and not infrequently possesses villous appendages. The consistence varies, being softer in recent cases, harder in older ones; it varies according as the hyperplasia affects the cellular or also the connective-tissue constituents. The appearance of a cut section of the spleen also varies. In some cases, there is simple hyperplasia of the splenic pulp: in others, there is hyperplasia of the follicles, which may attain the size of a walnut and have a round, elongated or caudate shape, or the trabeculæ may be hyperplastic. The organ sometimes presents a speckled, granite-like appearance. Hemorrhagic infarctions, and, if the disease has been preceded by intermittent fever, an unusual amount of pigment have also been found. Virchow mentions the formation of an abscess as a rare phenomenon.

Microscopical examination of the spleen does not disclose any abnormal constituents.

Chemical examinations of the organ have not furnished the same results. In 2,500 grams of splenic tissue, Salkowski and Stern found large amounts of peptonoid bodies, 0.288 hypoxanthin, 0.134 of other xanthin substances, 0.426 tyrosin, succinic acid doubtful, no uric acid. In 1,400 grams of splenic tissue, Boeckedahl and Landwehr found: peptones, 15.5 grams; lactic acid, 0.16; succinic acid, 0.029; xanthin, 0.548; leucin in large amounts; no hypoxanthin, uric acid, or tyrosin.

The liver is generally very large, and its weight may increase to 10 kilograms. The periportal glands are generally considerably enlarged. Upon section, the interlobular tissue is found more or less diffusely infiltrated with lymphomata, or we find very small nodules or firm nodes which have produced atrophy of the adjacent hepatic cells, so that in places only pigment detritus remains. If the liver is exposed to the air, the cut surface sometimes becomes covered with tyrosin crystals. On microscopical examination, the finer blood-vessels are not infrequently found to be almost entirely filled with white blood-globules, and their walls are often infiltrated with leucocytes. Cirrhosis of the liver is a rare complication of leukæmia.

According to Salkowski, chemical examination of the liver furnished the following results:

2,500 grams liver.

32	peptonoid substances.
1.718	tyrosin.
0.864	leucin.
0.2426	hypoxanthin.
0.538	other xanthin substances.
0.0852	succinic acid.

The stomach and intestines may also contain lymphomatous new-formations which form large prominences on the mucous membrane, and, in places, may surround the intestines like a ring. The lymphomata may start from the lymph follicles, or they may be heteroplastic. When they undergo ulceration, as in Friedreich's case, they may be mistaken for the lesions of typhoid fever.

The pancreas likewise may contain lymphomata.

The mesenteric and retroperitoneal glands may be very large. Virchow reported a case in which the pelvic glands were so large as to incarcerate, as it were, the pelvic organs.

The kidneys are often infiltrated with lymphomatous masses. They generally start from the surface, and are particularly numerous in the cortex. The kidneys sometimes contain uric-acid concretions. Under the microscope the accumulations of leukocytes are found to be especially abundant in the neighborhood of the vessels and glomeruli. A rare complication is waxy degeneration of the kidneys.

The suprarenal capsules are sometimes increased in size to such an extent by the development of lymphomata that they may undergo rupture.

The meninges and brain do not escape similar changes. Hemorrhages are frequent in these parts.

The larger retinal vessels have been found dilated and sinuous, the adventitia infiltrated with round cells; the smaller vessels present fatty degeneration, varicose dilatation and distention with round cells. The previously mentioned white patches consist in small part of sclerotic, hypertrophic nerve fibres and accumulations of granulo-fatty cells in the outer layers of the retina, in great part of an accumulation of leukocytes, mingled with red blood-globules. Similar changes are found in the choroid, and even in the iris.

The medulla of the bones always presents changes, to which Neumann has applied the terms lymphoid and pyoid. In the former, the medulla is gelatinous and red, and sometimes contains extravasations of blood; in the latter, the leucocytes have increased in number, and the medulla is opaque, grayish, pus-like. In both forms the fat cells disappear and are replaced by round cells. Waldstein observed nuclear fission in some of the large, mono-nuclear cells of the medulla. Neumann noticed profuse infiltration of the smaller arteries with round cells. Numerous Charcot-Neumann crystals are deposited on exposing the medulla to the air. The bone tissue is generally rarefied, although Henck observed osteo-sclerosis in one case.

No cases of leukæmia are known in which the blood-producing organs were intact.

IV. Diagnosis.—With the aid of the microscope the diagnosis of leukæmia is easy. The temporary increase of white blood-globules (leucocytosis) observed after eating, in fasting and marantic individuals, during pregnancy and infectious diseases does not reach such a high grade, and is not permanent.

V. Prognosis.—The prognosis is unfavorable. Cases of improvement or recovery are very rare, and even these are not undoubted.

VI. Treatment.—We may recommend nutritious, light food, and country air. Niemeyer obtained temporary effects from cold-water treatment. In addition, we may order iron, quinine, and cod-liver oil, and good effects are said to have been obtained from arsenic and phosphorus.

Many authors employ local treatment, i. e., directed against the splenic enlargement. Mosler recommends large, continued doses of quinine, alternating with eucalyptus and pyoctin. Others employ subcutaneous injections of ergotin or arsenic in the region of the spleen, or inject these substances directly into the spleen. According to Mosler, injections into the spleen should not be made unless the marasmus is not too far advanced, there is no tendency to hemorrhage, and the spleen is not too soft. An ice-bag should be applied over the spleen after each injection. Cold douches and compression, faradization, galvanization, and galvano-puncture of the spleen have also been employed.

Splenectomy is contra-indicated in this disease, inasmuch as all the patients died immediately after the operation.

2. *Pseudoleukæmia.*

(Hodgkin's Disease. *Adenia. Malignant lymphoma. Lymphosarcoma.*)

I. Etiology.—Pseudoleukæmia and leukæmia agree in their clinical and anatomical relations, except that, in the former disease, there is no increase of the white blood-globules in the blood.

Very little is known concerning its causes. Among those mentioned are intermittent fever, acquired and hereditary syphilis, scrofula, rickets, chronic diarrhœa, and alcoholic excesses. The disease sometimes follows pyæmia, chronic eczema, or dermo-mycosis, the adjacent glands being first affected, and the glandular hyperplasia then becoming general. In many cases no cause can be discovered.

It is more frequent in males, and from the ages of twenty to thirty years and fifty to sixty years. The laboring classes are more frequently attacked than the upper classes.

II. Anatomical Changes.—The anatomical changes, with the exception of the blood, are entirely similar to those of leukæmia. The enlarged lymphatic glands may be soft (hyperplasia of the round cells) or hard (also increase of the connective tissue). Ulceration, suppuration, or waxy degeneration are observed in rare cases.

III. Symptoms.—The disease generally begins with swelling of the cervical glands. Then other glands become swollen, either in the immediate vicinity or in remote parts (axilla, inguinal region). The process sometimes begins with general disturbances and lymphomatous degeneration of the tonsils. The scene may also open with enlargement of the spleen, followed by swelling of the internal and external lymphatic glands.

Anæmia soon develops, and is followed by all the symptoms mentioned under the head of leukæmia.

In the blood the microscope shows more or less diminution in the number of red blood-globules (poikilocytosis, microcytes, and numerous elementary granules).

The disease generally runs a more rapid course than leukæmia, but occasionally lasts several years. Febrile phenomena are sometimes noticeable, particularly when new glands become affected.

The pressure of the glands on adjacent organs may give rise to various complications, such as circumscribed œdema, paralysis of the recurrent laryngeal nerve, tracheal and bronchial stenosis, icterus, ascites, etc.

In some cases the disease is converted gradually into leukæmia. Some writers regard both diseases as identical, and believe that on account of the more rapid growth of the glands in pseudoleukæmia the lymph tracts are occluded, and the passage of white blood-globules into the blood thus prevented. Cohnheim's opinion, that pseudoleukæmia is an acute leukæmia in which death occurs before an excess of leucocytes enters the blood from the swollen glands, is disproved by the fact that the disease sometimes persists as such for several years.

IV. Diagnosis and Prognosis.—With the aid of the microscope, it is easy to diagnose the disease, and to distinguish it from leukæmia. The prognosis is as unfavorable as in the latter affection.

V. Treatment.—The causal indications (intermittent fever, syphilis, scrofula, rickets) should first be met. Cod-liver oil, potassium iodide, iron, and iodide of iron have been recommended in the treatment of the disease itself. The experience of Billroth and Czerny warrants the administration of arsenic internally, and in the form of injections into the glands. Trial may also be made of inunctions of green soap. Sapo virid., ʒ iv.–xij., is dissolved in a little lukewarm water, rubbed twice a week (for twenty minutes) into the back and limbs, and then washed off with water. M. Meyer caused enlarged glands to disappear by the action of the faradic current. The splenic enlargement is treated in the same way as in leukæmia.

3. *Melanæmia.*

I. Etiology.—In this disease, the blood contains granules of black or blackish pigment.

Intermittent fever, of severe types, is the sole known cause of the melanæmia. It is most frequent in the pernicious intermittents of the tropics, but occurs occasionally in the fever of our own latitude. The development of melanæmia depends on the severity of the infection, rather than upon the duration of the fever. Hence it is more frequent in certain epidemics.

II. Symptoms.—The chief feature is the appearance of dark pigment granules in the blood. These are free in the plasma, or inclosed in round cells, or in spindle-shaped cells which resemble the endothelium of the splenic veins, or they are contained in long, hyaline coagula. Finally, they may form long cylindrical structures, which sometimes appear fractured at one end or on both sides (vide Fig. 3). The granules are deep black, reddish-brown, or yellowish. The older, black particles resist for a long time the action of mineral acids and caustic alkalies; the younger ones grow pale very rapidly under their action.

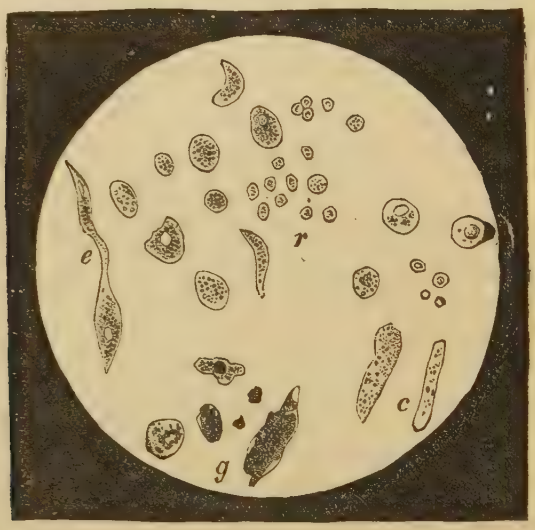
The majority of granules are inclosed in round cells, in which Mackenzie recently detected amœboid movements. The coagula which are sometimes found, consist of an albuminoid substance which dissolves in alkalies, and sets free the pigment granules. It is questionable whether the clots are precipitates of fibrin from the blood, or albuminoids derived from the destroyed red blood-globules. The pigment

masses, in the form of coagula, have been regarded as dislodged pigment emboli.

The pigment granules are round or irregularly angular, and vary in size. They sometimes exceed the dimensions of the blood-globules, and their edges are then often lighter in color, and laminated.

In some cases, the pigment disappears very rapidly after the cessation of an attack of fever, and reappears with the next attack. In other cases, it circulates in the blood for weeks and months. Melanæmia is sometimes the sole symptom, and is only recognizable after microscopic examination of the blood. It is generally associated with impoverishment of the blood in red blood-globules (oligocythæmia) and not infrequently with temporary increase of the white globules (leucocytosis). In some cases, other symptoms develop, the result in great part of the

Fig. 3.



Constituents of melanæmic blood. *r*, round cells, containing pigment; *e*, elongated cells with pigment (perhaps endothelium of the splenic veins); *g*, clots with pigment; *c*, cylindrical structures inclosing pigment. After Frerichs.

passage of the pigment into the capillaries of various regions of the body.

The skin assumes an ashen gray or grayish-yellow color.

Disturbances have also been observed on the part of the cerebral, intestinal, and renal functions, but it is doubtful whether the greater part is the effect of the intermittent fever or the secondary melanæmia. There is no doubt that the symptoms in question may occur in pernicious intermittent fever without co-existing melanæmia.

The most frequent cerebral symptoms are headache and vertigo; next coma or delirium; the most rare are convulsions and paralyses. These symptoms are said to result from occlusion of the cerebral capillaries by masses of pigment, and secondary extravasations of blood.

Similar processes in the domain of the portal vein are said to give rise to diarrhœa, enterorrhagia, peritonitic symptoms, and ascites.

Pigment emboli of the finer renal vessels cause anuria, albuminuria, and hæmaturia. Basch described a case in which the urine contained clumps of pigment, and similar ones were found in the blood.

III. Anatomical Changes.—The characteristic changes in the blood are recognizable in the dead body.

The largest amount of pigment is contained in the spleen; in rare cases, it is comparatively free, while the liver is very rich in pigment. The medulla of the bones, lymphatic glands, brain, kidneys, and skin also contain an abundance of pigment, and a smaller amount is also found in other organs (gastro-intestinal walls, pancreas, and lungs).

The spleen is generally enlarged as the result of the intermittent fever, and is soft or firm, according to the duration of the primary disease. Its appearance varies according to the amount of pigment which it contains. It is either speckled dark-brown or black, or has a diffuse black or slate color.

In the spleen, the pigment is inclosed, in great part, in round cells. Spindle cells, clots containing pigment granules, and free pigment are also found. The latter is most abundant in the blood spaces of the spleen itself, whence it passes into the surrounding pulp, particularly into the round cells. The Malpighian follicles are unaffected.

The periportal glands are not infrequently loaded with melanin. The large amount of pigment in the portal vein is evidently derived from the spleen. The liver is often swollen and not infrequently indurated. On section, it has a steel-gray color in places.

The interlobular branches of the portal vein are filled with pigment. The pigment then passes through the intralobular vessels to the central veins, and a part then passes into the inferior vena cava, right heart, lungs, and general arterial circulation. In this manner, pigment may also accumulate in the branches of the hepatic artery. A portion is probably carried by the amœboid round cells into the connective tissue surrounding the vessels. The liver cells are generally free, but Virchow found pigment in them.

The kidneys present black dots and streaks, the former corresponding to the glomeruli filled with pigment, the latter to the afferent vessels. Frerichs occasionally observed pigment in the urinary tubules.

In the brain, the cortex is affected almost exclusively, and assumes a chocolate-brown or graphite appearance. The medullary substance has a brilliant white color by contrast, but here and there it contains blackish streaks (pigment in the vessels). Small extravasations of blood are sometimes observed as the result of the pigment emboli.

The medulla of the bones has a brown, gray, or blackish color, and is usually poor in fat.

The pigment is undoubtedly the result of excessive destruction of red blood-globules. Since melanæmia is absent in other infectious diseases, an injurious influence upon the red blood-globules must be attributed to the malarial poison. The destruction of the red blood-globules is supposed by some to take place in the spleen, by others in the liver. According to Arnstein and Welsch, it takes place in the vessels themselves, and is deposited secondarily in the spleen and other organs.

IV. Diagnosis, Prognosis, Treatment.—The diagnosis can be easily and certainly made with the aid of the microscope. The prognosis is generally grave on account of the severity of the primary disease.

It should be treated prophylactically and causally with large doses of quinine (3 ss.–i. daily), continued for a long time; otherwise purely symptomatic treatment.

4. *Chlorosis.*

(*Chloræmia. Chloranæmia.*)

I. Etiology.—Chlorosis is an extremely frequent disease of the female sex. It is very rare in men, and some writers deny, though improperly, its occurrence in the male sex. The affected males are generally slender and of a feminine type.

The disease generally develops at the period of puberty (fourteenth to twenty-fourth years). It is not so very infrequent in children, and in exceptional cases develops at the age of thirty years or later.

Chlorosis is an exquisitely hereditary and congenital disease. Virchow showed that it is the result of imperfect development of the vascular apparatus (hypoplasia), which may or may not be associated with feebleness of the entire body and hypoplasia of the sexual apparatus.

Chlorosis is often hereditary in families which also suffer from phthisis, cancer, and nervous diseases.

In many cases, certain auxiliary factors produce complete development of the latent germs of the disease. Such factors may also produce the disease in the absence of hereditary and congenital predisposition to chlorosis (acquired form). These include psychical conditions: excessive study, grief, homesickness, etc. In others, physical factors are at fault, for example, sedentary habits, confinement in close, poorly ventilated rooms, working in factories, insufficient nourishment, etc. It sometimes follows vital losses, such as those connected with child-bed, lactation, or masturbation.

The constitution is not such an important factor as is generally believed, since delicate and feeble girls are not the only ones affected. The patients almost always suffer from menstrual disturbances, but we must be on our guard against mistaking cause and effect. Niemeyer states that all girls who menstruate at the twelfth or thirteenth year, before the development of the breasts and pubis, become affected with chlorosis.

The frequency of the disease is constantly increasing. This is partly the result of the perverse bodily and mental training incident to modern civilization.

II. Symptoms.—The symptoms generally develop gradually. They occasionally begin immediately after the first menstrual period. The patients generally seek medical advice on account of subjective complaints: general feebleness and lack of desire to work, drowsiness, rheumatoid pains, dyspnœa, palpitation, gastric disturbances, etc. In other patients, the menses first become irregular, scanty, and painful, or cease entirely. In rarer cases the individual feels quite well, but has a pale, miserable appearance.

The pale color of the skin is one of the most constant and earliest symptoms. It appears earliest and most markedly on the lobe of the ear. The cheeks also lose their red color and the mucous membranes (conjunctiva, lips, gums, and remainder of buccal mucous membrane) become pale-red or yellowish-red. In some patients the entire face has a sallow or greenish-yellow color, and in brunettes it is sometimes a dirty pale gray.

The sclera is often bluish-white, and the subconjunctival fat pale yellow.

The amount of pigment sometimes diminishes in the skin, and even in the hairs, either in patches or diffusely. The nails generally have a deathly pale color on account of the diminished redness of the nail bed.

The integument is generally dry, has very little tendency to perspire, and often desquamates.

Certain chlorotics, however, have a blooming complexion, as the result of dilatation of the subcutaneous vessels in the face. Others blush deeply with every bodily or emotional excitement, evidently from hyper-excitability of the vaso-motors.

The panniculus adiposus is often unusually well developed. If it disappears very rapidly, a suspicion of the existence of some wasting disease (generally phthisis) must be aroused.

Œdema develops occasionally; it is generally very slight, affects the ankles or lids, and disappears at night. In rare cases more extensive and permanent œdema of the legs is observed. The œdema is owing to the fact that the changes in the blood disturb the nutrition of the walls of the vessels, and render them abnormally permeable.

If the finger is pricked with a needle, the blood generally flows freely, so that it does not seem to be diminished in this disease. But, as a rule, it is pale red, serous, and watery. Under the microscope, the red blood-globules often seem, even without careful measurement, to be diminished in number. They are often very pale and exhibit but little tendency to assume the nummular arrangement. Blood-globules of very large dimensions often alternate with extremely small ones. Changes in the shape are also very frequent. The globules are constricted, pear-shaped, or entirely irregular (poikilocytosis). In not a few cases the white blood-globules are diminished in number. In some cases there are numerous protoplasm granules, occasionally aggregated into large heaps.

Accurate count has shown that the number of red blood-globules is sometimes unchanged, sometimes diminished. In one case Laacher found 2,440,000 red blood-globules in one cub. mm. (normally 4,430,000).

Even the amount of hæmoglobin may be unchanged, though, as a rule, it is diminished (sixty-seven per cent of the normal, on the average, in twenty-four cases examined by Laacher).

Duncan noticed that if the red globules are placed in a solution of sodium chloride, they lose their coloring matter more readily than the blood-globules of healthy individuals.

The amount of iron in the blood is diminished. The blood serum may be unchanged, or the proportion of albumin may be increased or diminished.

The temperature of the body is almost always unchanged. There are occasionally slight elevations in temperature (39.8° in the rectum is not infrequent, according to Mollière, and may be relieved by quinine).

The majority of patients complain of a subjective feeling of coldness. The pulse is usually soft, accelerated, and its rapidity often varies greatly in consequence of bodily or mental exertion.

As a rule, the patients exhibit a disinclination to mental or bodily work. They have a tired expression of the face, and often sleep during the day, while at night they toss about restlessly in bed.

The mood is irritable, capricious, and tearful. The patients often complain of shortness of breath which is increased by movement. They are not infrequently hoarse, and in such cases the laryngoscope shows

striking pallor and dryness of the laryngeal tissues. Catarrhs of the deeper air passages are not infrequent, and their development is evidently favored by the diminished powers of resistance of the system.

The breasts become very flabby and diminish in size; not infrequently they contain hard spots which may occupy the entire gland.

The majority of patients are annoyed by palpitation, which develops spontaneously or after mental or bodily effort. The vigorous action of the heart is often visible over several intercostal spaces. The right, rarely the left, ventricle is often dilated (anæmic dilatation). Systolic murmurs are often audible over one or several, or even all the valves. If the heart's action is very vigorous, the second pulmonary sound is often intensified temporarily, thus arousing the suspicion of mitral insufficiency. The differentiation between the two conditions sometimes requires prolonged observation.

Dilatation of the right ventricle may be the result of nutritive disturbances of the heart muscle, to which the thin-walled right ventricle is most apt to yield. The murmurs are probably dependent on the same cause, inasmuch as the improperly nourished heart muscle is incapable of regular vibrations during contraction. The systolic sound is always heard in addition to the murmur. The murmur is rarely musical, nor does it often give rise to thrill. It is heard most frequently over the pulmonary valve, next over the mitral and tricuspids, rarely over the aortic valves.

Accidental diastolic murmurs are heard in rare cases.

In not a few cases, the carotids pulsate vigorously, and the cardiac systolic sound is often converted, in these vessels, into a murmur. A short, low systolic sound may be heard over the smaller arteries, and on pressure is converted into a compression murmur; on increased pressure, into a pressure sound.

The bruit de diable is often heard over the bulb of the internal jugulars (between the sternal and clavicular portions of the sterno-mastoid, immediately above the sterno-clavicular joint); but this is also found, at times, in healthy individuals. The murmur is sometimes conveyed across the manubrium along the right border of the sternum.

The bruit de diable may be sighing, roaring, sawing, or whistling. It is not infrequently felt as a thrill. It increases in the erect position, in deep inspiration and slight rotation of the head towards the opposite side, and is generally louder on the right side than on the left. This is owing to the fact that the right internal jugular runs a more vertical course to the heart, so that a more vigorous whirl is formed within its bulb than in the left jugular. Compression of the peripheral portion of the vein causes disappearance of the murmur, on account of interruption to the blood supply. The murmur may be so loud that it is heard by the patients as an annoying roaring in the head.

Similar murmurs are sometimes heard over the crural vein, immediately below Poupart's ligament, but these increase during expiration; in rare cases, they are heard over the subclavian or even the facial vein.

The cervical veins are generally but slightly filled. They sometimes manifest a negative venous pulsation.

Chlorotics very often suffer from goitre, but I have never been able to hear a murmur over the tumor. It disappears as the primary chlorosis is relieved. It is also said that the patients may suffer from exophthalmus.

Persistent fœtor ex ore is observed not infrequently in chlorotics. Anorexia is a frequent symptom, but sometimes we notice bulimia, in-

creased thirst, or a desire for peculiar articles (pica). Many complain of gastric distention, violent pain in the stomach, eructations, and vomiting. Constipation is quite a constant symptom.

The urine is generally light-colored, watery, and of low specific gravity. The urea and uric acid may be diminished. The urine often contains traces of albumin, but no casts.

Disturbances of menstruation are very common. In the majority of cases, the menses remain entirely absent. In others, they occur regularly, but are attended with violent pain, last only a few hours, and the fluid has more of a mucoid than bloody appearance. The menses may also be very irregular, and finally they may be very profuse.

Complications of a functional or organic character are observed very often. The former include nervous disturbances, such as spinal irritation, neurasthenia, cephalalgia, neuralgias, more rarely spasms and paralyses. The latter are particularly apt to develop when, as is not uncommon, chlorosis is followed by hysteria.

Among the various forms of neuralgia, particular attention is merited by gastralgia, since chlorosis creates a predisposition to round ulcer of the stomach.

Chlorotics often suffer from leucorrhœa, and examination with the speculum may disclose erosions and ulcerations of the vaginal mucous membrane and portio vaginalis.

Virchow emphasizes the tendency of chlorotics to endocarditic changes. Cases of venous thrombosis have also been described, perhaps as the result of fatty degeneration of the endothelium.

Repeated epistaxis is often observed.

Finally, chlorosis creates a certain predisposition to pulmonary phthisis.

In three cases, Gowers observed optic neuritis and neuro-retinitis, which improved after the administration of iron. Pallor of the fundus is a common symptom of chlorosis.

The duration and course of the disease depend upon its causes. If dependent on hereditary and congenital influences, frequent relapses must be expected, and certain symptoms may persist permanently. In other cases, it may be relieved in a few weeks.

III. Anatomical Changes.—Death occurs only from intercurrent diseases, so that very little opportunity is afforded for post-mortem examinations.

Increased development of the adipose tissue is often noticeable, not alone in the panniculus adiposus, but also the subepicardial and omental fat. The internal organs are generally pale.

The pale, often flabby heart is unusually small, but the right ventricle is often dilated in comparison with the other cavities. The endocardium is delicate, transparent, and bluish-white. In places it contains slightly elevated, light-yellow patches, which correspond to fatty degeneration. Streaks of fatty degeneration are also often found in the heaat muscle.

The aorta is likewise unusually small and delicate, and occasionally is not larger than the crural artery of a healthy individual. Its walls are often very delicate and distensible. The intima has a bluish appearance and presents yellowish elevations. The microscope shows fatty degeneration in the intima and tunica media. The intercostal arteries are very

often given off irregularly from the aorta. Similar changes are observed in other arteries.

There may be defective development of the sexual apparatus, though this is not constant.

Fatty degeneration may develop in the liver, kidneys, pancreas, and the glandular cells of the gastro-intestinal tract.

Nothing is known with certainty concerning the nature of the disease. We regard it as a primary affection of the blood-producing organs, which gives rise to the production of a diminished number of red blood-globules containing a diminished amount of hæmoglobin. We confess, however, that we possess no positive proof in support of this hypothesis.

IV. Diagnosis.—The diagnosis is easy, but it must be remembered that chlorosis sometimes occurs in individuals with a ruddy complexion.

The disease is distinguished from secondary anæmias by the fact that chlorosis is a primary condition. Under certain circumstances, however—for example, latent phthisis and cancer—secondary anæmia may be mistaken for primary sclerosis. We should be on our guard, therefore, when chlorosis develops in a woman with a phthisical family history, and when it is attended by rapid emaciation and night sweats.

It is distinguished from progressive pernicious anæmia by its amenability to treatment, the absence of prolonged febrile movement and of retinal hemorrhages.

Chlorosis is differentiated from chronic nephritis, in those cases in which œdema and slight albuminuria are present, by the absence of casts in the urine.

V. Prognosis.—The prognosis is always good as regards danger to life. The symptoms can be rapidly relieved in the majority of cases, but it is often impossible to prevent relapses.

VI. Treatment.—The treatment consists mainly of rational bodily and mental regimen. But although fresh air is very necessary for the patients, we should not, at the start recommend long walks, since these often do more harm than good. Exercise in the open air should never be carried to such an extent as to tire the patient. Cold rubbings are useful in order to make the body more resistant. A change of air is generally beneficial. In some cases such simple measures, attended with removal of the etiological factors, will suffice to effect a cure.

Among the medicinal agents the most important are the ferruginous preparations.

We agree with those writers who recommend long-continued and large doses of iron.

Almost every practitioner has a favorite iron preparation. Our own preference is for Blaud's pills.

The various preparations may be arranged in the following order, with regard to readiness of assimilation: ferrum redactum, ferrum lacticum, ferrum pulveratum (gr. iss. three hours after meals), tinct. ferri pomata, tinct. ferri acetici, tinct. ferri chlorid. (30 drops t. i. d.).

In some individuals, the mildest preparations produce gastric symptoms. In such cases, iron has been used subcutaneously, especially ferrum oxydat. dialysat., ferrum pyrophosphoric c. ammonio citrico (1 : 5), ferrum pyrophosphoric. c. natrio citrico (1 : 6), one syringeful subcutaneously. The solutions should be freshly prepared, since fungi are apt to form in them, and often lead to the development of an abscess after injection.

Iron is absorbed to a very slight extent in the gastro-intestinal tract, so that some writers think it acts by producing hyperæmia of the mucous membrane, and thus favoring the absorption of food.

Iron waters and baths are also employed in many cases. Ziemssen reports favorable results from the administration of hæmoglobin lozenges.

If the patient also exhibits evidences of scrofula, we should order iodine in combination with iron, for example, ferrum iodat. saccharat. (gr. iss. every two hours), syrup. ferri iodid. (℥ ss. t. i. d.), etc. Cod-liver oil should also be administered (℥ ss. morning and evening). In a number of cases I have obtained remarkably good results from the use of Kissingen water, together with baths.

If gastric symptoms are present from the start, we must be very careful in the use of ferruginous preparations. Gastric digestion may be improved by the administration of hydrochloric acid (gtt. v. in half a wineglassful of lukewarm water one-half hour after dinner and supper). If gastro-intestinal peristalsis is inactive, we may order the bitters, for example, tinct. chinæ comp. (℥ i. t. i. d.), elix. aurant. comp. (℥ i. t. i. d.), strychnin. nitricum (gr. ¾, pulv. althææ, q. s. ut ft. pil. No. xv. D. S. One pill t. i. d.), etc.

In some cases, the symptoms disappear rapidly after a happy marriage, but in other cases women become profoundly chlorotic after marriage.

5. *Progressive Pernicious Anæmia.*

(*Essential Pernicious Anæmia. Idiopathic Anæmia. Anæmatosis.*)

I. Etiology.—The disease is characterized by increasing impoverishment of the blood, which increases almost uninterruptedly to a fatal termination.

In the minority of cases, it develops without any demonstrable cause (idiopathic or primary form); in others it follows certain injurious influences (deuteropathic or symptomatic form). But we would relegate to the category of pernicious anæmia only those cases in which there is a striking disproportion between the cause and effect. Hence a sort of predisposition is necessary in order that progressive pernicious anæmia may develop.

The secondary form may result from mental strain; excessive bodily labor may act in the same way. In some cases, it is the result of living in unhealthy rooms, working in overcrowded factories, and insufficient food. The disease sometimes develops during pregnancy or after confinement. It occasionally follows diarrhœa, vomiting, repeated epistaxis, vital losses, and typhoid fever.

It is most frequent from the twentieth to sixtieth years; in very rare cases, it occurs during childhood.

The geographical distribution of the disease varies remarkably. It is especially frequent in Switzerland, particularly in Zurich and its vicinity. Even in Zurich its frequency varies greatly at different times. Numerous cases have been observed in England, France, and Sweden, while Italy, Spain, and Russia escape almost entirely.

II. Symptoms.—The clinical history depends entirely on the intensity of the impoverishment of the blood.

The disease generally begins gradually. The patients grow tired, and perspire after slight exertion; they are short of breath, suffer from palpitation, perhaps complain of dizziness, and grow paler day by day. Finally they are unable to leave the bed.

Among the manifest symptoms, the chief attention is attracted by the intense pallor of the face and mucous membranes. The sclera is not infrequently icteric in color.

Jaundice is a rare symptom. In a few cases, the skin assumed a gray or brownish color, as in Addison's disease.

The cutaneous secretions are diminished, so that the skin appears dry. The nutrition of the hairs is sometimes impaired. They become brittle, destitute of gloss, and fall out. In one case, I observed nutritive changes in the nails; they were thickened and fissured, and crumbled off at the free edges.

Hemorrhages under the skin are not infrequent. They generally occur earliest and most abundantly upon the lower limbs, are usually about the size of a pin's head (petechiæ), rarely they cover a large surface (ecchymoses) or have a swollen appearance (ecchymomata). They are sometimes shaped like stripes (vibices), generally as the result of the pressure of folds of the clothing.

Punctate hemorrhages may also occur upon the mucous membranes (scleral conjunctiva and buccal mucous membrane).

Œdema is a frequent symptom. It does not always appear first in the lower limbs, but occasionally in the face. At a later period, also, the face may be swollen into a shapeless mass, while the œdema of the limbs is very slight. Œdema of the conjunctiva (chemosis) is frequent. At the beginning of the disease, the œdema may be temporary. At a later period, it becomes permanent, and is associated with slight serous accumulations in the serous cavities. This symptom is probably the result of nutritive disturbances in the walls of the vessels.

The subcutaneous adipose tissue is often unusually abundant; in other cases, it is more or less atrophied. The physique is generally slight, but robust persons may also be affected. Some of the bones may be tender on pressure. In rarer cases, there are pains in the muscles.

The bodily temperature may be normal during the entire course of the disease. In other cases there are elevations of temperature (40° C. or more), of a continuous, remittent, or irregular type.

The pulse is generally soft and accelerated.

The subjective complaints of the patient consist chiefly of a feeling of great weakness. Palpitation, obscuration of vision, dizziness, nausea, and syncope often set in as soon as the patient attempts to pass from the recumbent to the erect position. Many also appear to suffer from mental weakness. They lie in an apathetic manner, as if half asleep, sometimes mutter to themselves, and answer questions after a long interval, as if the meaning of the question were only understood gradually. Some complain of obstinate insomnia, anxiety, a feeling of constriction in the chest, etc.

The sensorium is sometimes unaffected almost to the last moment. In others, there is increasing somnolence with a dreamy condition, in which life is gradually extinguished. In still others, delirium and maniacal attacks are observed, and they must be carefully guarded in order to prevent them doing injury to themselves or those about them.

There is often persistent insomnia. The patients are sleepy in the day, but at night they toss to and fro, moan, and talk to themselves.

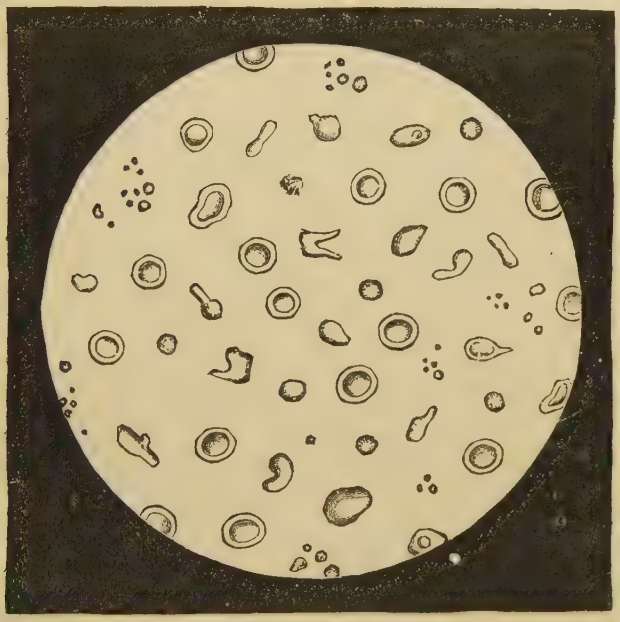
The respiratory organs are unaffected. Attacks of dyspnœa may develop spontaneously or after excitement, and are the effect of anæmia.

There is also a tendency to epistaxis, which may be repeated, increases the anæmia, and occasionally is directly dangerous to life.

The circulatoy organs rarely escape functional disturbances. In many, attacks of palpitation occur spontaneously or after slight excitement. The movements of the heart are then unusually vigorous, but the subjective symptoms may also exist without increased heart's action. Not infrequently there is slight dilatation of the heart, usually the right side alone, occasionally also the left. The first heart sound is often converted into a systolic murmur, which may be heard over one or all of the cardiac orifices. In rare cases, we hear diastolic murmurs of a purely accidental character.

The carotids generally pulsate vigorously, and systolic murmurs are

Fig. 4.



Blood in progressive pernicious anæmia.
Poikilocytosis and a few spherical microcytes. Enlarged 600 times.

often heard over them. A short arterial sound may also be heard in peripheral arteries (brachial, axillary, radial).

The bruit de diable over the jugular vein is a constant symptom. True or negative venous pulse is often visible in the external jugulars. The bruit de diable may also be heard in the crural vein, but, unlike that in the jugulars, it grows feebler during inspiration, stronger during expiration.

If the finger is pricked with a needle, the blood flows freely. As a rule, it is light colored, sometimes of an amber yellow. Coagulation may take place very slowly. The white blood-globules are generally very scanty. The so-called protoplasm granules (elementary granules, hæmatoblasts) are present in small numbers. The number of red blood-

globules is very much diminished. Kjellberg found in one case only 571,000 in 1 cub. mm. (normally 4,000,000 to 5,000,000). The red blood-globules are pale, irregular in shape, and vary in size. They are elliptical, provided with prolongations, pear-shaped, etc. (vide Fig. 4) (poikilocytosis). As a rule, their dimensions are increased (8–9 μ instead of 7.6 μ). Not a few attain a diameter of 15 μ (giant blood-globules). In addition, there are others which are unusually small (microcytes), and these are either umbilicated or spherical. The former are intensely red and glistening, and are rather rare. Their diameter generally varies from 3–4 μ. Very fine drops of a hæmoglobin color are sometimes found. The red blood-globules are generally isolated, rarely arranged in columns. Stellate shapes are rarely observed.

In one case the blood had a reddish-brown or a coffee-like color, in another it was unusually dark. Stricker describes brownish blood-globules. The hæmoglobin has been found separated from the stroma and accumulated in drops. Pilz mentions amœboid movements of the red blood-globules. A few nucleated red globules have also been observed.

The changes described may also occur in other forms of anæmia. Frankenhaeuser described, in the blood of pregnant women suffering from progressive pernicions anæmia, spherical movable bodies with a swinging lash, which are supposed to have entered the blood from the liver and to constitute a certain stage of development of leptothrix (?). In three of my cases these bodies were also noticeable, but I could arrive at no conclusion with regard to their nature.

In one case Quincke found that the blood amounted to 5%, in another to 4.34% of the weight of the body (normally 8%).

In one case Fraenkel found that 100 parts of the blood, four days before death, contained 11.57 solid matters, of which 1.81 were nitrogen (15.66% nitrogen in the dried blood). Healthy blood gave the following figures: in 100 parts 20.24 solid matters, 3.27% nitrogen, 16.17% nitrogen in the dried blood.

Disturbances of the digestive organs are observed almost constantly. Hemorrhages from the gums and small ulcers upon the buccal mucous membrane have been observed in several cases. The majority of patients complain of anorexia and fœtor ex ore. Sometimes there is insatiable bulimia and the feeling of thirst is occasionally increased. Complaint is often made of pain and pressure in the gastric region, a burning feeling, eructations. Vomiting is frequent, and hæmatemesis is observed occasionally. The latter may continue for a long time, and rapidly exhaust the patient. Diarrhœa, sometimes of a bloody character, is a frequent symptom.

The liver and spleen are generally normal in size; in advanced cases, they are sometimes slightly enlarged. The liver may be very tender on pressure.

The urine is passed in large quantities, but there may be striking variations on successive days. It is sometimes very dark, the specific gravity is normal, the reaction always acid.

Chemical examination of the urine furnishes varying results. The urea is sometimes diminished, sometimes increased; sodium chloride is almost always diminished. Increase of the amount of indican has been noticed.

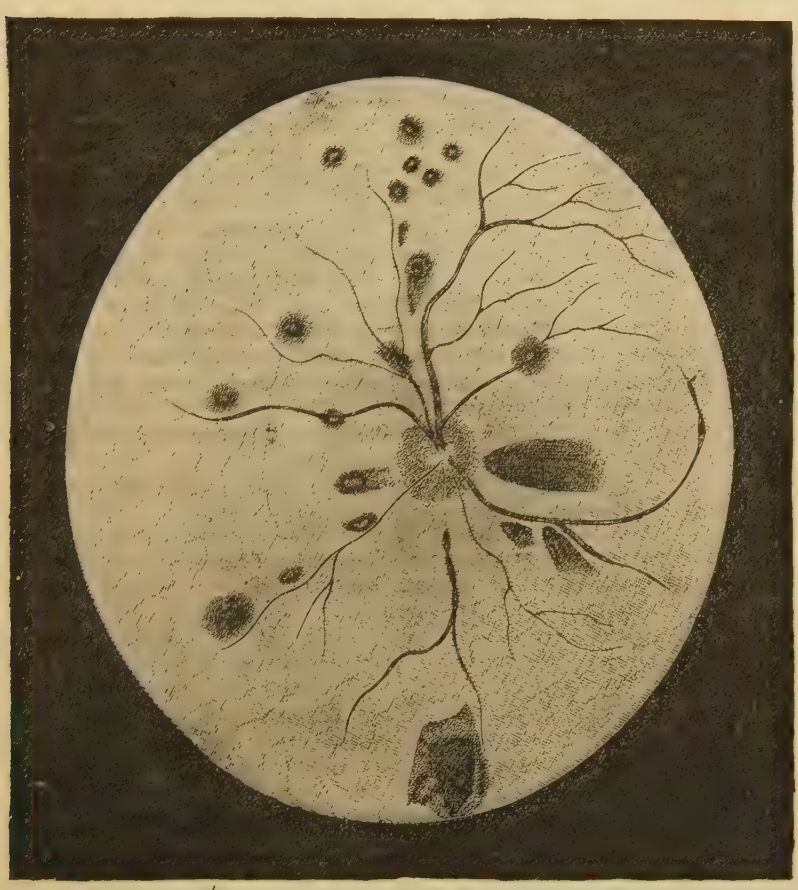
Albuminuria is rare; Laacher observed peptonuria. Hoffmann found lactic acid in the urine and an increased amount of kreatinin. Hæmaturia has been observed occasionally.

The nervous system is very often affected. Twitchings, paretic and paralytic conditions, and paræsthesiæ are not unusual, but generally only temporary.

The nerves of special sense may be involved. The patients suddenly become deaf, lose smell and taste, or complain of abnormal sensations in some of the nerves of special sense. Sudden blindness develops occasionally, and is sometimes the result of retinal hemorrhages.

Vision is often intact despite the almost constant occurrence of retinal changes. The latter consist mainly of hemorrhages, which are sometimes present in astonishing numbers (vide Fig. 5). They vary greatly in size, but are sometimes almost as large as the optic disc.

FIG. 5.



Retinal changes in progressive pernicious anæmia.
Light centre in the hemorrhages. After Quincke.

They are most abundant in the neighborhood of the papilla. They are generally streaked, and often radiate towards the papilla. The recent ones are ruby-red, the older ones brownish-red, the latter not infrequently containing a light-yellow centre. Large numbers sometimes appear suddenly in the course of a single day, but they may be absorbed in a relatively short time (two to three weeks).

The retina sometimes contains yellow patches similar to those found in Bright's disease.

Œdema and symptoms of stasis in the retina and papilla are observed much less frequently. The retina assumes a veiled, reddish-gray appearance, the borders of the disk are indistinct, the papilla prominent, and the retinal veins distended and sinuous, while the arteries are very narrow.

The disease sometimes lasts only a few weeks. In other cases, life is prolonged for months, sometimes even for years. The subacute and chronic cases often present remissions and exacerbations, but, as a rule, the disease terminates fatally.

A few cases of the conversion of progressive pernicious anæmia into other diseases have been reported. For example, it has been followed by the symptoms of myelogenic leukæmia, or of sarcomatosis of the bones. In one of my cases it was followed by a beginning cancer of the pylorus. As the anæmia had existed for a year, and the cancer was just beginning, it would be irrational to regard the anæmia as the result of the cancer. Another of my cases was complicated during the course of the disease by lympho-sarcoma of the mesenteric and retro-peritoneal glands.

In some cases death follows the gradual extinction of all the functions, in others a rise of temperature occurs for days or hours before death. But sometimes the temperature falls to a very marked extent (25.8° C.). The cutaneous perspiration sometimes has a cadaverous odor for a few hours before death.

III. Anatomical Changes.—The integument remains extremely pale. The panniculus adiposus may be unusually developed, and there is sometimes an increase of fat in the internal organs, particularly beneath the epicardium and in the mesentery. The muscles are pale, and sometimes very dry.

Under the microscope, the muscles are generally found to be intact. Berger found colloid degeneration of these organs. Mueller describes fatty degeneration of the diaphragm and intercostal muscles. E. Fraenkel noticed a large amount of yellow and brown pigment in the ocular muscles: the muscular fibres were cloudy and granular.

The serous cavities contain moderate amounts of transudation of an amber yellow. It is occasionally sanguinolent or icteric.

Hemorrhages are noticed upon the skin, in the muscles, the mucous membranes, serous membranes, and the interstitial tissue of many organs. They are generally small, often punctate, rarely of large size. They are sometimes so large, however, as to give rise during life to hæmoptysis, hæmaturia, or hæmatemesis, although no bleeding vessel can be found after death.

The internal organs are unusually pale, the cavities of the heart are almost empty, or contain small quantities of watery blood, which is either entirely fluid or deposits scanty thin clots. The latter sometimes have an icteric color.

In one case, the blood in the dead body had an acid reaction. According to Quincke, the specific gravity is 1028.2 (normally 1055).

The heart is sometimes unusually small, sometimes it is dilated, particularly the right ventricle. Hypertrophic changes may also be noticed. The heart muscle is generally pale and brittle. If the epicardium is thin, yellow patches sometimes can be seen shining through, and these are seen still better under the endocardium. They are most abundant

in the left heart, particularly in the papillary muscles of the mitral valves. They are often so numerous that the heart muscle has a butter-yellow, speckled, marbled appearance, and are found to correspond to marked fatty degeneration of the muscular fibres. The fat is generally arranged in coarse granules; one part of a fibre may be affected, while adjacent parts are intact. Nuclear proliferation is absent, but there are often small interstitial hemorrhages. These appearances are rarely absent in progressive pernicious anæmia.

The endocardium is thin and transparent, and occasionally presents hemorrhages, slight fatty degeneration and atheromatous changes. The valvular apparatus is always intact.

The aorta is generally normal. In a few cases it has been found to be narrow, or to present fatty degeneration and atheroma.

The respiratory organs are little changed. Small hemorrhages into the lungs have been observed, and occasionally œdema of the glottis.

The spleen is generally normal in size, but slight enlargement is not very rare. In the latter event, the parenchyma is firm and tough. Hemorrhages into the organ have also been reported.

In one case, Leber found a large amount of leucin and tyrosin in the spleen, liver, lungs, and pancreas. Attention has been called recently to the large amount of iron in the spleen.

The liver is generally normal in size. In rare cases, it is slightly enlarged. It is often pale, at other times the central veins are distended. Hemorrhages are observed not infrequently. The gall-bladder is often filled with dark bile. Pepper observed ecchymoses in the mucous membrane of the gall-bladder.

The hepatic cells are often in a condition of fatty degeneration. In one case, Mueller and Winge observed the development of adenoid tissue with an accumulation of round cells.

A large amount of iron has been found in the liver.

The gastro-intestinal mucous membrane often presents œdematous swelling and extravasations of blood. Enlargement of the intestinal lymph follicles and fatty degeneration of the glandular epithelium have also been observed.

Juergens and Sasaki described fatty degeneration and atrophy of the nerve plexus of the intestines, but these changes are probably secondary.

The mesenteric glands are often swollen, hyperæmic in places, and speckled with blood. In one case, all the glands were swollen and red, and the lymphatics were dilated and contained bloody lymph.

The pancreas may be very large and congested, with hemorrhages into the interstitial tissue. The glandular epithelium is in a condition of fatty degeneration.

The kidneys are generally very pale, and the epithelium of the tubules is not infrequently fatty. In a few cases, there is slight increase of the interstitial tissue with accumulation of round cells. Thickening of the Malpighian capsules, and fatty degeneration of the blood-vessels have also been noticed. Hemorrhages have been observed upon the mucous membrane of the urinary passages and sexual organs.

Extravasations occur frequently in the meninges, particularly on the inner surface of the dura mater.

The brain almost always contains numerous capillary hemorrhages, generally in the white matter. Certain vessels are sometimes in a condition of fatty degeneration. In one case, Schumann found spindle-shaped and ampullary dilatations of the vessels. The brain and spinal cord are very pale.

Changes in the sympathetic system (proliferation of the interstitial connective tissue, atrophy of the nerve fibres and ganglion cells) have been described in a number of cases, but Lubimoff has shown that we must be very careful in our interpretation of such appearances.

In my own cases the peripheral nerves were intact.

The retina contains hemorrhages and yellow patches; the optic papilla is sometimes œdematous and swollen.

In recent cases, the hemorrhages consist solely of red blood-globules. In older cases, granular disintegration begins in the centre of the extravasation, and extends peripherally; in such cases the ophthalmoscope shows a light yellow centre. In rare cases white patches are produced by the presence of white blood-globules in the centre of the extravasations. In one case, Krukenburg noticed varicose nerve fibres.

The hemorrhages are generally situated free. In a few cases, the blood is situated in the adventitious lymph space.

The retinal vessels not infrequently contain spindle-shaped and ampullary dilatations.

In many cases the medulla of the bones is unchanged. In other cases it contains hemorrhages, or the fat tissue disappears and is replaced by red lymphoid medulla.

In addition to a large number of spherical red blood-globules, the medulla contains numerous nucleated red blood-globules (so-called transitional forms). It often contains a large number of cells which inclose blood-globules.

The majority of writers believe that progressive pernicious anæmia is a disease of the hæmatopoietic organs which leads to insufficient formation of blood. Perhaps the imperfectly formed red blood-globules sometimes undergo unusually rapid destruction. Whether the spherical blood-globules are poorly developed or are approaching destruction, remains doubtful.

All other anatomical and clinical symptoms depend upon the impoverishment of the blood. The fatty degeneration of the heart and glandular epithelium, and the abundant development of the panniculus adiposus are probably owing to the fact that, on account of the impoverishment of the oxygen-carriers in the blood, the albuminoids of the tissues not alone are decomposed freely into urea-forming and fat-forming substances, but that the latter remain in situ and are not oxidized into carbonic acid and water.

The hemorrhages are sometimes the direct result of the impoverishment of the blood, rather than of fatty degeneration and rupture of the vascular walls. The nutrition of the vessels seems to be disturbed in such a way that the red-globules readily pass through by diapedesis.

The changes in the medulla of the bones are also secondary and anæmic in character, and E. Neumann has shown that they develop after other anæmic and cachectic conditions.

IV. Diagnosis.—The diagnosis is by no means easy, particularly at the onset of the disease. Among other things, latent cancer may lead to error. The disease is readily distinguished from chlorosis, because the

latter occurs almost exclusively in women at the period of puberty, is apyrexial, and disappears rapidly under the use of iron. The occurrence of fever may lead to the diagnosis of typhoid fever, endocarditis, or meningitis, but the further course of the disease will clear up the diagnosis. Finally, atrophy of the peptic glands may present the symptoms of progressive pernicious anæmia, but the former disease is rare, and its independent existence is even questionable.

V. Prognosis.—The majority of patients are inevitably doomed. According to some writers, intermissions may last for months and years, but finally a fatal relapse supervenes.

VI. Treatment.—If the disease is recognized early, we should order a change of air and nourishing food, especially a milk diet. The patients must avoid all bodily and mental exertion.

Caution must be exercised in the administration of iron, since it is not tolerated by many patients. As in chlorosis, we prefer Blaud's pills; if these are not tolerated, the ethereal tinctures of iron may be employed. Some authors recommend phosphorus, and particularly arsenic.

When the anæmia becomes excessive, it is said that transfusion of blood (perhaps sodium chloride is preferable) sometimes produces good, and even permanent effects.

6. *Purpura Simplex.*

I. Symptoms.—In this disease, hemorrhages occur into the skin. They are generally round, as large as a pin's head, on the average; at first discrete, later confluent in places. They are especially abundant upon the legs and back of the hands. The hemorrhages occur as simple patches (purpura maculosa) or as papular elevations (purpura papulosa). Here and there are noticed wheals which do not itch, and generally become hemorrhagic. At first the patches are blood-red, then brownish-red, then green, and finally yellow.

The exanthem may have been noticed accidentally, or it may have been preceded by slight fever, gastric disturbances and a feeling of malaise. New crops often appear, particularly if the patients walk a good deal. The average duration is ten to fourteen days.

II. Etiology.—In many cases no cause can be discovered. It sometimes occurs in anæmic, phthisical, or scrofulous individuals, after prolonged diseases or shortly before the appearance of the menses.

III. Prognosis, Treatment.—The prognosis is always favorable. The treatment consists merely of rest and nourishing diet, perhaps iron in anæmic individuals.

7. *Purpura Rheumatica* (*Peliosis Rheumatica*).

I. Symptoms and Diagnosis.—This disease consists of purpura of the skin, and painful swelling of the joints.

It is often, though not constantly, preceded by prodromata. The patients feel weak and depressed, and have slight fever. In a few days, they complain of rheumatic muscular pains, and of pains in some of the joints. The ankle and knee joints are affected most frequently, sometimes certain of the other joints, particularly the elbows. Not infrequently there is slight swelling of the joints.

Soon after, or coincidently with the pains in the joints, patches of purpura appear on the skin. They occur most abundantly and early on

the legs, later the trunk and upper limbs may be affected. The extensor surfaces are the favorite site, in some cases in the vicinity of the diseased joints. According to their age, the patches are dark, almost black-red, brownish-red, green, or yellow. They do not grow pale on pressure, and a few are elevated into papules. The legs are often œdematous, and œdema of the eyelids is also observed not infrequently. Urticaria may be present in certain parts, and either disappears or is replaced by hemorrhages.

The pains in the joints generally subside with the onset of the purpura. The patches are gradually absorbed in five to ten days. In rare cases, the purpura is the first, the joint changes a later symptom.

The disease may terminate in one to two weeks, but relapses often occur, so that it sometimes drags along for months and years. Fever may or may not be present. Kaltenbach noticed apyrexia in the morning, elevation of temperature in the early part of the afternoon, and gradual defervescence at night. Bohn described fever of a tertian type. Anæmic symptoms develop if the disease is protracted. Enlargement of the spleen has been repeatedly observed.

Hemorrhages into the mucous membranes are generally absent, but hæmaturia, hemorrhage of the gums, followed by gangrene, and bloody discharges from the genitalia have been described.

II. Etiology.—The disease is most frequent in men from the age of fifteen to thirty years. It is rare in childhood, and has never been observed in infancy. I observed it very often in Berlin, more frequently in autumn and winter, and sometimes almost in the form of an epidemic. In women, the symptoms sometimes begin shortly before the menstrual period. It is claimed that a predisposition to the disease is created by anæmia, articular rheumatism, malaria, phthisis, and heart disease. I recently treated a case in which the disease followed a gonorrhœa.

III. Anatomical Changes have been described by Leuthold and Traube in a patient who died from a complicating tubercular pyopneumothorax. The joints contained an abundance of clear synovia; the synovial membrane was injected and contained old hemorrhages. Hemorrhages were also found in the extensors of the knee joint.

IV. Prognosis and Treatment.—The prognosis is almost always favorable, except in those cases in which hemorrhages occur into the mucous membranes.

The treatment is similar to that of morbus maculosis Werlhofii.

6. *Purpura Hæmorrhagica.*

(*Morbus Maculosus Werlhofii.*)

I. Etiology.—The disease manifests itself by spontaneous hemorrhages, not alone into the external integument, but also into the mucous membranes and internal organs.

Females are attacked more frequently than males. The disease is most common from the age of fifteen to twenty years, and is extremely rare in infancy. Cases have been reported at the age of five months, and at birth. Delicate, poorly nourished individuals are predisposed to the disease, though robust persons are not entirely exempt. This disease is relatively frequent in northern countries and at the sea shore, and is more common in winter than in summer.

In many cases, no cause can be ascertained; in others, it is attributed to cold, exposure, damp dwellings, and insufficient nourishment, so that the disease is sometimes endemic in barracks, foundling asylums, and boarding-schools.

The symptoms of the disease sometimes appear during convalescence from severe infectious diseases, particularly typhoid and intermittent fever, and also during pregnancy or child-bed.

Dohrn reports a case in which a pregnant woman, suffering from purpura hemorrhagica, gave birth to a child who presented evidences of the same disease. This was evidently owing to the influence of the same causes upon the blood and blood-vessels of mother and fœtus.

A few cases of toxic purpura have been reported, for example, after the inhalation of sewer gas, after the ingestion of pork, and in a man who was buried in a well for ninety-six hours.

II. Symptoms.—The disease begins suddenly, or it is preceded by prodromata. The latter consist of anorexia, general malaise, vomiting, vertigo, and slight fever. They last for a few hours or several days.

The first visible changes are the cutaneous hemorrhages. These appear first on the legs, later on the trunk and upper limbs. In many cases, the face escapes. The extensor surfaces are affected more severely than the flexor surfaces.

The majority of the extravasations are as large as the point of a needle or a pin's head; a few vary from the size of a pea to that of a bean. In rare cases, we find ecchymomata or vibices; the latter as the result of pressure of the bedding or clothing. The extravasations are sometimes so closely aggregated that the skin appears to be diffusely hemorrhagic. With increasing age, the color of the extravasations changes to brownish-red, blue, green, and yellow.

Among the rare complications is elevation of the epidermis into vesicles, evidently from the accumulation of blood between the epidermis and rete Malpighii. Urticaria is occasionally observed. Suppuration or gangrene of the hemorrhagic parts is a rare event. Still rarer is the escape of blood in fine drops upon the surface of the epidermis. Hemorrhages can sometimes be produced voluntarily by pressure upon the skin.

Hemorrhages upon the mucous membranes appear at the same time, or soon afterwards. They are most frequent upon the nasal mucous membrane, and may cause more or less violent epistaxis. Hemorrhages into the lips, cheeks, and gums are not infrequent; and those into the gums may give rise to very violent hemorrhage, although the parts are not swollen or loosened. In a few cases, vesicles form upon the buccal mucous membrane. Hæmatemesis or enterorrhagia indicates hemorrhage from the gastro-intestinal mucous membrane. These symptoms are sometimes very violent, and may even terminate in perforation-peritonitis. This is explained by the fact that an infarction of the intestinal mucous membrane may cause necrosis of the part, and finally rupture into the peritoneal cavity. Marked hæmaturia, metrorrhagia, and hæmoptysis have been observed. Hemorrhages beneath the conjunctiva, into the retina, choroid, and even the sclera have been repeatedly described. In older retinal hemorrhages, the centre becomes light yellow as absorption occurs, and the focus may disappear entirely in a few weeks. There may be considerable retinal hemorrhage without visual

disturbance. Epileptiform attacks and paralysis are sometimes observed as the result of meningeal and cerebral hemorrhages.

After extensive hemorrhages, the blood may grow lighter in color. Under such circumstances, the white blood-globules may be increased in number, the red globules diminished (in one case, 900,000 in one cub. mm.). In two cases, Penzoldt observed microcytes. Several authors mention absent or diminished coagulability of the blood.

In some cases, the general condition is very little affected. In others, there is fever; the patients are pale and miserable, complain of weakness, and, if the anæmia increases, suffer from albuminuria, palpitation, dizziness, and syncope. Death may result from recurrent or uncontrollable hemorrhages. The joints are sometimes slightly swollen and painful.

The average duration of the disease is two to six weeks. In some cases, death occurs in a few hours or days after the appearance of the first hemorrhages; in others, the disease lasts for several months.

Relapses have been observed in a number of instances. In Rohlfs' case, twelve relapses occurred in twelve years.

The sequelæ include paralysis as the result of cerebral hemorrhage, and in one case, diabetes mellitus (perhaps from hemorrhage into the medulla oblongata). Fagge states that he has seen six cases in which sarcoma developed in various organs.

III. Anatomical Changes.—The majority of deaths are the result of anæmia, so that the internal organs are very pale. The cutaneous hemorrhages remain visible after death. The intermuscular connective tissue, fasciæ, tendons, and periosteum are generally intact, but extravasations are found not infrequently in the serous membranes and internal organs. The suprarenal capsules have been found entirely filled with blood, and the intestinal mucous membrane sometimes contains bloody infiltrations of considerable size. Transudations into the serous cavities are not infrequently hemorrhagic. Extravasations have been found in the medulla of the bones, the endocardium, intima of the vessels, and the neurilemma. The spleen is often enlarged, and sometimes contains infarctions. Hindenlang described pigment infiltration of the lymphatic glands.

Wilson found amyloid degeneration of the capillaries in the vicinity of the petechiæ. Variot attempted to show that the hemorrhages are the result of diapedesis, not of rhexis, but this does not hold good of all cases. For example, Hayem reported a case in which the white blood-globules were increased in number, and had given rise to hemorrhages, by forming thrombi in the finer arteries. Stroganow discovered infiltration of the intima of the aorta, vena cava, and hepatic veins with red blood-globules which seem to have passed, by diapedesis, directly from the lumen of the vessels into the tunica intima. In Hindenlang's case, the pigment in the glands formed clumps, was evidently produced by a transformation of blood pigment, and consisted of hydrated ferric oxide.

Nothing is known concerning the nature of the disease, but it seems to be a primary affection of the blood, with secondary injurious effects upon the walls of the vessels.

IV. Diagnosis.—The diagnosis is easy.

Purpura simplex is confined chiefly to the external integument; at all events, it presents no free hemorrhages.

In peliosis rheumatica, the joint changes are prominent, and free hemorrhages do not occur.

Scurvy shows the specific affection of the gums.

Hæmophilia is an hereditary or a congenital, permanent disease.

Acute exanthemata of an hemorrhagic character are attended with high fever and specific cutaneous changes.

V. Prognosis.—As a rule, the disease runs a favorable course. Sudden onset, high fever, and profuse hemorrhages are grave symptoms. Death occurs with relative frequency in pregnant and puerperal women, because abortion and uncontrollable uterine hemorrhage may be produced.

VI. Treatment.—The patients should be kept constantly in bed. The diet should be nutritious, and stimulating articles, such as coffee, tea, and alcoholics, should be interdicted. Thirst should be relieved by sulphuric-acid lemonade. The bowels should be evacuated daily. If signs of weakness appear, we may order the following:

℞ Decoct. cort. chinæ ..... ℥ vi.
Acid. sulphur. dil. ..... ʒ i.
Syr. simp. ..... ℥ ss.
M. D. S. One tablespoonful every two hours.

Otherwise, purely symptomatic treatment.

9. *Scorbutus.*

(*Scurvy.*)

I. Etiology.—Scurvy is clinically related to purpura hemorrhagica and purpura rheumatica. Hemorrhages occur upon the skin and mucous membranes in this disease, and are associated with a tendency to inflammations.

Scurvy is an inanition process, which may result from various, sometimes diametrically opposed causes.

The most important cause is poor nourishment.

In some cases, scurvy is the result of insufficient food, so that it is often observed in prisons, during famine, long sieges, long trips at sea, etc.

It is not infrequently the result of the ingestion of spoiled articles of food, but this cause is often associated with the former one. Good drinking-water is an important feature in such cases.

In a third group of cases, the quantity and quality of the articles of diet are sufficiently good, but their combination is defective. Most important is abstinence from fresh vegetables, particularly potatoes. Thus epidemics of scurvy have been observed in Ireland and England as the result of failure of the potato crop. On the other hand, there is no more certain and rapid means of relieving scurvy than by giving plenty of fresh vegetables. In like manner, abstinence from fresh meat may produe scurvy. A very fruitful cause of the disease is the excessive ingestion of salted or corned beef. Some epidemics have been attributed to an insufficiency of fat in the diet.

Living in damp, poorly ventilated, and overcrowded dwellings, and exposure to wet also act as causes of scurvy.

Epidemics due to these causes have been observed in prisons, barracks, orphan asylums, arctic explorations, etc.

It is evident, from the foregoing considerations, that the geographical position and meteorological conditions influence the development of the disease. It is often observed in northern latitudes and in rainy, cold districts. Epidemics are more frequent in winter and spring than in summer and autumn.

Bodily and mental strain must also be included among the causes of scurvy. It has been observed repeatedly among sailors and besieged, when they were required to perform increased work (perhaps also, because the food then proved insufficient). Sometimes, however, the hard-working individuals escaped, while the drones were attacked by the disease. It is also said that, under such circumstances, courage and a hopeful disposition antagonized the spread of scurvy, while the hopeless ones and cowards fell victims to the malady. Homesickness (nostalgia) has also been regarded as an important causal agent.

Scurvy is more frequent in men than in women, because the former are more exposed to its exciting causes.

For the same reason, the disease generally occurs in middle life.

A predisposition to scurvy is sometimes congenital or acquired. Under like circumstances, feeble individuals are affected more quickly and severely than robust ones. Drunkards are affected more readily than others, and scurvy sometimes occurs in individuals who take little else beyond alcoholics. It is also more apt to occur in patients who have suffered from malaria, dysentery, typhoid fever, or syphilis.

Scurvy generally occurs in epidemics. The losses of life from this cause were formerly enormous, so that in some sieges a larger number fell victims to scurvy than to the weapons of the enemy.

But even at the present time, the disease is endemic in certain regions, for example, in Russia and Roumania.

II. Symptoms.—Scurvy rarely develops suddenly, and such cases generally run an acute and often a pernicious course. As a rule, it is preceded by prodromata which sometimes last only a few days, but often one to two weeks or even longer.

The patients gradually lose their healthy color, the skin becomes dry, fissured, and scaly. The face becomes sallow, the lips livid, the eyes dull and sunken, and dark-brown patches of pigment are sometimes observed in the face.

The patients are generally very depressed and even desperate. They lose appetite, more rarely manifest boulimia or a desire for sour, piquant articles of food, and become weaker and weaker. Dyspnœa and palpitation become noticeable on slight exertion, and complaint is made not infrequently of headache, a feeling of pressure in the head, and attacks of syncope.

As a rule, inflammatory changes in the gums constitute the first manifest symptoms, but they sometimes remain absent or are preceded by other scorbutic symptoms. The affection of the gums generally begins at the anterior surface of the incisors, and then extends internally and laterally. The changes are not observed in places where the teeth are absent, but they crawl along the stumps of teeth.

The veins at the free edge of the gums are first distended, the gums become swollen and their tissue loose, and the color becomes bluish; slight contact produces pain and more or less severe hemorrhage. The parts situated between adjacent teeth are particularly swollen. The gums become loose and may proliferate to such an extent as to come in contact above the teeth. The teeth may be loosened and fall out, either in an intact or carious condition. The swelling of the gums may be so intense that the vessels are compressed, the tissue becomes necrotic and is converted into a brownish or blackish pulp.

The inflammation of the gums is so much less intense the greater the distance from the free edge of the teeth, and it generally ceases en-

tirely at the base. The mucous membrane of the lips and cheeks almost always remains intact. Pharyngitis has been observed in a few cases, and Pinder observed excrescences and ulcers on the posterior surface of the pharynx.

The inflammation of the gums is attributed to mechanical causes. Scurvy produces a tendency to inflammation in the most varied tissues, and the constant mechanical irritation of the gums during mastication sets up the inflammation in this part.

The patients generally complain of pain during eating, but apart from this they are often free from suffering. There is often a pestilential fœtor ex ore. In some patients the secretion of saliva is increased, so that a sanguinolent fetid fluid flows almost constantly from the mouth.

If recovery occurs, a complete restitution of the gums may take place. In some cases, however, a firm, cicatrix-like tissue forms and persists for life.

Hemorrhages into the skin, subcutaneous tissue and muscles appear coincidently with or soon after the changes in the gums, rarely at an earlier period.

The cutaneous hemorrhages generally appear as petechiæ which vary from the size of a flea-bite to that of a nail. They appear earliest and most abundantly on the legs, especially the extensor surfaces; the trunk or limbs are often affected at a later period, but the face generally escapes. Traumatic influences (pressure, blows, prolonged walking) not infrequently give rise to hemorrhages. They are often very numerous in the vicinity of old cicatrices. The limb is sometimes thickly covered with them.

The first changes often appear around a hair follicle, so that the blood-vessels surrounding the latter are evidently the starting-point of the hemorrhage. The hair not infrequently becomes dry and fibrillated, and falls out. The accumulation of blood is sometimes so considerable that the skin is raised in flat or pointed papules (lichen et acne scorbuticus). The blood collects occasionally between the epidermis and rete Malpighii, raising the former into vesicles (herpes et pemphigus scorbuticus). In pemphigus, the vesicle may burst, leaving an ulcerated base, covered by a bloody crust. After removal of the latter, a readily bleeding surface is exposed; it is covered with abundant granulations, has little tendency to heal, and sometimes furnishes a foul-looking, fœtid secretion. Free extravasations upon the skin have been observed in a few cases.

Hemorrhages into the subcutaneous connective tissue may be very extensive, and sometimes surround the entire circumference of a limb. They develop acutely or slowly, and, in the former event, are generally attended with pain and elevation of temperature. As a rule, the overlying skin can be moved very little or not at all, feels doughy, is often painful on pressure, and its temperature is increased. Such hemorrhages are seen with special frequency around the tendo Achillis and hamstrings, and are often the result of traumatic or mechanical causes. They may disappear without leaving any residua, or they are followed by sclerotic thickenings of the skin. Or adhesions form to underlying parts and impede the mobility of the limbs. This may terminate in the production of pes varo-equinus or other deformity, or of false ankylosis of the knee joint. The underlying muscles may also undergo atrophy

as the result of pressure. Inflammation and suppuration sometimes set in, the skin is perforated, and a chocolate-colored, sometimes foul-smelling mass is discharged. This terminates in ulcerations similar to those following cutaneous hemorrhages. Hemorrhages next to or beneath the nails may give rise to inflammation, sometimes to loss of the nails (onychia and paronychia scorbutica).

Muscle hemorrhages occur most frequently into the calves, extensors of the thighs, buttocks, and abdominal muscles. They are so much more painful the more rapidly they develop. Suppuration and perforation through the skin may ensue, or sclerotic thickenings are left over with contracture and deformity of the limbs, or atrophy and muscular weakness develop.

Hemorrhages from the mucous membranes (epistaxis, hæmatemesis, enterorrhagia, hæmaturia, more rarely metrorrhagia and hæmoptysis) are much less frequent, but they occasionally prove fatal.

Painful swelling of the joints develops in some cases. The fluid in the joints may be purely serous or hemorrhagic. The process is sometimes followed by suppuration, erosion, deformity of the ends of the bones, and true ankylosis.

Hemorrhages and inflammations may also occur in the serous cavities. The inflammations are generally hemorrhagic in character, and most frequently involve the pleura or pericardium, more rarely the peritoneum. They often develop with extreme rapidity, and produce profound anæmia or place the patient in danger of suffocation from compression of the lungs or of paralysis of the heart. Some authors have observed rapid absorption of exuded fluid. Meningeal hemorrhages also occur and are characterized by pain, paræsthesia, spasms, contractures, paralyses, and apoplectiform attacks.

Subperiosteal and epiphyseal hemorrhages are rare events in scurvy. The former chiefly involve the anterior surface of the tibia, but may also occur on other bones (scapula, lower jaw, hard palate). The accumulated blood forms a painful swelling, but generally undergoes absorption. The epiphyseal hemorrhages are relatively most frequent on the costal cartilages, where they cause separation of the ribs from the cartilages, so that the free ends of the ribs sink inwards.

Scorbutic changes are observed not infrequently in the eye. Conjunctival hemorrhages and inflammations may set in, likewise hemorrhages into the anterior chamber or hemorrhagic chorioiditis. The peculiar form of keratitis (generally bilateral) with subsequent panophthalmitis, which is seen after disease of the trigeminus (vide Vol. III., page 68), has also been observed. Hemeralopia occurs with relative frequency, as a prodrome, during the development of the manifest symptoms or as a sequel.

As the manifest symptoms of scurvy gradually develop, the general condition grows worse. The appearance of the patient becomes cachectic, and the panniculus adiposus and muscles waste away. In some cases, however, the general nutrition remains intact for a very long time. Fever may be absent or it is slight and irregular. Considerable elevation of temperature is generally the result of abscess formation. The heart presents anæmic dilatation and systolic murmurs, and the spleen is sometimes enlarged. There is occasionally diarrhœa which may assume a dysenteric character. In a number of cases a connection has been observed between scurvy and true dysentery. The urine varies in amount, is generally acid, and its specific gravity diminished. It often

contains albumin, but this does not justify the diagnosis of nephritis, which is a rare complication.

The amount of urea is generally diminished; Simon observed increased excretion of uric acid. In a number of cases the amount of potash salts was increased. Grocco mentions peptonuria.

Examinations of the blood do not furnish uniform results. The following changes have been observed at times: diminution or abolition of coagulability, increased alkalinity, diminution of the potassium and iron and increase of sodium chloride, increase or diminution of the amount of albumin.

The most frequent complication is fibrinous pneumonia, which probably takes its origin in many cases from hemorrhagic infarctions, and may terminate in gangrene. Scorbutics are sometimes attacked by other infectious diseases (variola, typhoid and relapsing fevers, septic endocarditis).

The disease generally runs a chronic or subacute, more rarely acute course. The subacute cases terminate in four to eight weeks, the chronic cases in as many months.

A fatal termination is not rare. It may be the result of the increasing weakness, or of excessive exudation into the pleural and pericardial cavities, of pneumonia or profuse hemorrhages, or finally it may follow symptoms of a septic condition.

In favorable cases, convalescence sometimes lasts a long time. A marked tendency to relapses is left over.

III. Anatomical Changes.—Rigor mortis is generally poorly marked, and livores mortis are very numerous. There is also a tendency to rapid cadaveric changes. The cutaneous hemorrhages are recognizable after death. The subcutaneous and intermuscular hemorrhages not infrequently contain clots, and manifest changes in the connective tissue which have terminated in gelatinous or firm connective-tissue proliferations and thickenings. Beneath subperiosteal hemorrhages, the superficial layers of bone are reddened and sometimes necrotic. Softening of pre-existing callus or absence of the formation of callus in recent fractures is said to have been observed in some cases. Uskow described lymphoid changes in the medulla of the bones. The joints often contain serous or sanguinolent effusions, hemorrhages into the cartilages and synovial membrane, erosion of the cartilages, and sometimes an accumulation of pus.

The serous cavities often contain pure fluid blood, or clots mixed with inflammatory products. More or less extensive hemorrhages are often found in the subserous connective tissue.

The blood may be cherry red and thin; its quantity may be very small, so that the viscera are anæmic and not infrequently fatty, though at the same time they often contain extravasations.

The heart is flabby, brittle, pale brown in color, and fatty in places. Subepicardial hemorrhages are frequent, subendocardial hemorrhages are rarer. Endocarditic changes are found occasionally. Marantic thrombi are sometimes present in the heart, particularly in the right auricle.

Subepithelial hemorrhages have developed not infrequently in the bronchial mucous membrane. In the lungs, we often find œdema (sometimes hemorrhagic), or pneumonic or gangrenous changes, or hemorrhagic infarctions following extravasations or emboli (the latter secondary to cardiac thrombi).

The spleen is often enlarged and very soft, and not infrequently contains hemorrhagic infarctions.

The gastro-intestinal mucous membrane frequently presents bloody suffusions, also follicular ulcerations and necrotic (diphtheritic) changes.

Hemorrhages and fatty degeneration have been observed in the liver.

The kidneys are generally intact. Bloody suffusions are frequent on the mucous membrane of the urinary passages and genital organs.

Leven found fatty degeneration of the muscles, earliest in those which are most used, viz., the heart, muscles of the back, thighs, arms, etc. The liver and kidneys were affected next in order of frequency. In the capillaries and small arteries of the gums and intestinal mucous membrane, the endothelium has been found swollen, so that cells lying opposite one another came in contact and produced occlusion. On the central aspect of such places, the endothelium cells were separated from one another, and the red blood-globules had passed through the interstices into the surrounding tissues.

No corpuscular or chemical changes have been found in the blood or other organs, so that nothing is known concerning the nature of the disease.

Many assume that scurvy is an infectious disease, which is at the same time miasmatic and contagious. We only know that certain cases develop under bad hygienic conditions, *i. e.*, under circumstances in which other infectious diseases also develop. Contagion from man to man has not been proven.

Some authors attribute scurvy to an excess of sodium chloride in the blood, but no valid proofs have been offered in support of this theory.

This is also true of the potassium theory of scurvy. Garrod shows that fresh meat and vegetables are distinguished by the larger amount of carbonate of potash and vegetable salts of potash from those articles of diet whose prolonged ingestion gives rise to scurvy (in the blood the vegetable salts of potash are converted into the carbonate). Now, if there is insufficient carbonate of potash in the food, the blood and tissues grow poor in potash salts, and scurvy is the result. This may also happen in certain cases despite the presence of sufficient potash salts in the food, for example, after diarrhœa, or after perverse nutrition of the tissues as the result of bodily or mental strain.

IV. Diagnosis.—The symptoms are so characteristic that the diagnosis is easy. Special weight should be attached to the affection of the gums.

V. Prognosis.—The prognosis is not always good, because it is often impossible to bring the patients rapidly under other hygienic and dietetic surroundings.

VI. Treatment.—Prophylaxis has been attended with brilliant results. Ships and beleaguered towns must be supplied plentifully with good water, fresh meat and vegetables, particularly potatoes and sauerkraut. Fresh fruits are also useful, especially oranges and lemons. Overcrowding of dwellings should be avoided, etc.

After scurvy has made its appearance, we should first endeavor to meet the causal indications. If the patient is placed in a well-ventilated room, and supplied with fresh meat, vegetables, beer or wine, the symptoms often disappear rapidly without medication. Especially useful is the fresh juice (℥ ij.–℥ vi.) daily of certain cruciferae, such as cress, radish, sorrel, sauerkraut, various kinds of cabbage, dandelion, etc. Some recommend beer yeast in doses of ℥ v.–x. daily.

The drugs to be recommended are, the vegetable salts of potash (potassium citrate, bitartrate, acetate, binoxalate).

If signs of anæmia are prominent, we may order iron, quinine, and bitters. Giommi reports a successful result after transfusion.

Prominent symptoms often require treatment. When the gums are affected, the mouth should be gargled, after each meal, with chlorate of

potash (ʒ i. : ℥ v.) or acetate of alumina (gr. xv.: ʒ iij.) Ulcers on the gums and exuberant proliferations are touched with the solid stick. Drastics must be avoided in scurvy, since they are apt to produce dangerous intestinal hemorrhage.

Hæmophilia.

(*Hæmatophilia. Hæmorrhophilia.*)

ETIOLOGY. — "Bleeders" are individuals in whom severe hemorrhages, which often terminate fatally, develop spontaneously or after very slight causes.

In the majority of cases, the disease is hereditary, and can sometimes be traced for many generations. The predisposition is generally inherited by males, and is very rare in females.

The heredity is either direct or indirect. In the former, hæmophilia occurs in every generation; in the latter, some generations may escape.

Women are the most potent factors in heredity, although, as a rule, they remain free from the disease. If a bleeder marries a woman who comes of a healthy family, the children, as a rule, remain free from hæmophilia. But if a healthy man marries a woman who, although herself healthy, comes of a family of bleeders, the children are almost always bleeders.

Some of the male descendants escape the disease, and it is not probable that such individuals will beget hæmophilic children, if married to a healthy woman. According to Wachsmuth, families of bleeders are very prolific, their average number of children being nine, those of healthy families only five.

Allied to hereditary hæmophilia is the congenital form. This form includes those cases in which children are bleeders, although the parents came of healthy families. Very little is known concerning the cases of congenital hæmophilia. Among those mentioned are: marriage between blood relations, phthisis, scrofula, rheumatism or gout in the parents, and fright during pregnancy.

Some writers assume the spontaneous development of hæmophilia in later life. This is asserted with regard to those cases in which the symptoms were not manifested during childhood, but developed at a later period. It might be claimed, however, that accidental exciting causes were absent during childhood.

The chief contingent of the patients is furnished by Germany, next follow England, France, and North America. Among 210 families of bleeders, 94 lived in Germany, 52 in Great Britain, and 23 in North America. Among 780 cases, 717 were males, 63 females. A predisposition to hæmophilia has been attributed to the Anglo-Germanic family and the Caucasian race, but Heymann has recently reported a case in a Mohammedan family of Java.

II. SYMPTOMS.—The symptoms are sometimes observed accidentally, as when the patient has a violent hemorrhage, either spontaneously or after slight causes. Such facts may possess a medico-legal interest. For example, Wunderlich reports a case in which a boy, mildly punished by his teacher, was found covered with numerous extravasations. Legal steps were about to be taken to secure the punishment of the teacher, when it was discovered that the boy was a bleeder.

A similar discovery is sometimes made accidentally by surgeons when making an operation upon a patient.

In women, hæmophilia is sometimes masked behind profuse, long-

continued menstruation. Kehrer has also shown that fatal hemorrhage after delivery may set in in such cases, and has recommended that premature labor be induced.

The occurrence of rheumatoid pains, neuralgia, especially of the teeth, swelling and pain in the joints in the children of bleeders, indicate that the former are also bleeders. This becomes almost a certainty if profuse epistaxis is frequent.

In some cases, it is said that the muscular pains are followed by contractures and atrophy of the muscles.

The children sometimes fall a victim to the disease immediately after birth, as the result of uncontrollable hemorrhage after tying the umbilical cord. But, on the one hand, this does not occur very often, and, on the other hand, does not always depend on hæmophilia.

The first symptoms of the disease appear most frequently at the period of first dentition, in rare cases not until the period of puberty is fully developed. In one case the first manifestations appeared at the age of twenty-five years. Many bleeders die before the tenth year, and they rarely attain old age. The hæmophilic disposition sometimes diminishes with increasing years, but rarely disappears before the twenty-fifth year.

The spontaneous hemorrhages are sometimes preceded by prodromata, or, properly speaking, prodromal molimina, which consist of palpitation, rush of blood to the head, dizziness, ringing in the ears, etc. Certain individuals feel relieved after the cessation of the hemorrhage.

The spontaneous hemorrhages occur most frequently as epistaxis, next in order of frequency beneath the skin, more rarely from the kidneys, air passages, intestines, or genitals. Articular hemorrhages are not infrequent. The joints are distended, fluctuating, and extremely painful, and the affection may terminate in erosion of the ends of the bones, ankylosis, and suppuration. Suppuration, gangrene, and perforation of the skin have also been observed as the result of the subcutaneous extravasations, which sometimes attain astonishingly large dimensions. The mass discharged often has a chocolate color, and is mixed with gangrenous shreds. It must be remembered that the blood in subcutaneous hæmatomas remains fluid for a very long time, and that incautious opening may give rise to fatal hemorrhage. This has also been observed after spontaneous rupture.

In a few cases, spontaneous hemorrhages have been observed upon the peritoneum, meninges, and into the brain.

Traumatic hemorrhages are often produced by accidents. Cases have been reported in which the pricking of the gums by a tooth-pick or biting the tongue produced a fatal hemorrhage. Rupture of the hymen has also been known to terminate fatally. Uncontrollable hemorrhage has been observed with special frequency after the extraction of teeth. Fatal hemorrhage has been observed hitherto in ten cases of circumcision. Vaccination is relatively harmless, but leech bites and cupping are dangerous. Small wounds are sometimes more dangerous than larger ones, and Fordyce succeeded in checking the hemorrhage in one case by enlarging the wound with the knife. The same wound will produce varying grades of hemorrhage in the same individual at different times. The hemorrhage is almost always capillary. The blood flows from the wound as from a wet sponge, and no bleeding vessel can be discovered.

At first the blood has a normal color, but after the bleeding has lasted for days, it assumes a serous and watery character.

Microscopical and chemical examination furnishes negative results. The number of red blood-globules is sometimes increased. Hérard states that the organic constituents of coagula are diminished, the salts increased.

The hemorrhages are sometimes so profuse that death ensues in a few hours. In other cases, they last for days and weeks. The enormous amounts of blood lost, and the rapidity with which the patients are restored, are often astonishing. Syncope following anæmia of the brain sometimes checks the hemorrhage, inasmuch as it depresses the blood pressure.

Albuminuria is noticed occasionally at the height of the hemorrhage. There are no other constant urinary changes. If the anæmia is excessive, œdema may develop, together with anæmic changes in the heart (dilatation, systolic murmurs). Febrile conditions are sometimes observed.

It has been claimed that bleeders are characterized by slight physique, blond hair, blue eyes, superficial vessels, and a tendency to blushing, but there are many exceptions. Kunze observed premature grayness of the hairs, Legg and Sedgwick found multiple nævi in bleeders.

III. Anatomical Changes.—No constant changes have been found in hæmophilia. Virchow noticed smallness of the heart, narrowness of the vessels, and thinness of their walls. The left ventricle was occasionally hypertrophic, or fatty degeneration was noticed in the intima of the vessels. Recent enlargement of the spleen has also been described.

Microscopical changes in the cutaneous vessels have been described in a few cases, but they were probably mere coincidences. Birch-Hirschfeld noticed enlargement of the endothelium cells in the capillaries and transitional vessels, swelling of their nuclei, and granular deposits in their protoplasm; silver preparations showed unusual irregularity of the endothelium. Kidd described increase of the endothelium, dropsical swelling of the muscular coat, and proliferation of its nuclei, in the finer vessels of the subcutaneous connective tissue and in the muscles. Legg was unable to find these changes.

Nothing is known with certainty concerning the nature of the disease. Immermann attaches chief importance to the narrowness and thin walls of the vessels, and to increase in the amount of blood, which occasionally seeks an outlet. In addition to the thinness of the walls of the vessels, Cohnheim assumes impoverishment of the blood in red blood-globules (unproven) and a secondary tendency to hemorrhages. The following hypothesis seems to me to be the most plausible: diminution of the number of white blood-globules (Assmann), hence slow production and diminished resistance of coagula (Lossen), injurious effect of the changed blood on the vessels, with increased tendency to diapedesis and rhexis. Why the white globules are diminished in number is unexplained.

IV. Diagnosis.—The recognition of the disease is not difficult if manifest symptoms are present. It is distinguished from scurvy by the fact that it is unattended with inflammation and proliferation of the gums, and is not a temporary condition. The latter circumstance also distinguishes it from morbus maculosus Werlhofii and purpura. The differentiation between hæmophilic and bacteritic hemorrhage in the new-born is based upon the presence or absence of bacteria in the blood.

V. Prognosis.—The prognosis is always grave, so much more the greater the opportunity for sustaining injuries offered by the occupation of the patient.

VI. Treatment.—Prophylaxis must first be directed against the

spread of hereditary hæmophilia, by prohibiting marriage in families in which the disease is hereditary.

This alone, if carried into effect, would not extirpate the disease, since it sometimes develops primarly (congenitally) from unknown causes. Inasmuch as experience teaches that a number of children in a family suffer from the disease, it would be wise to recommend that the parents endeavor to prevent an increase in their family.

Bleeders should be protected, as much as possible, against injuries. They should be exempt from conscription as soldiers. Their diet should not include stimulating substances, such as alcoholics, tea, or coffee. If a hemorrhage occurs, it must be treated according to surgical principles, by rest, elevation of the bleeding part, prolonged compression, the actual cautery, and sutures. Not much can be expected from internal hæmostatics.

Surgical operations must be avoided in bleeders. Vaccination alone is devoid of danger. Circumcision should not be performed in bleeders.

The internal remedies recommended in hæmophilia include iron, ergotin, acetate of lead, and laxatives (in congestive conditions).

If anæmia becomes excessive as the result of hemorrhage, transfusion with sodium chloride solution may be resorted to.

Rheumatoid and neuralgic complaints, and non-hemorrhagic swelling of the joints must be treated symptomatically.

PART II.

DISEASES OF THE SPLEEN.

Acute Enlargement of the Spleen.

(*Acute Splenic Tumor.*)

I. Etiology.—Acute splenic tumor is a rapid enlargement of the organ which only lasts a short time. It is merely a symptom of certain primary affections.

We distinguish four varieties, viz., traumatic and embolic acute enlargement, the spleen of acute stasis and of acute infection.

Traumatic enlargement of the spleen is not very frequent. It is the result of a blow, fall upon the spleen, etc. Anatomically, it consists of increased amount of blood, often of extravasations of blood, swelling, and proliferative processes in the cells of the splenic pulp.

Embolic acute enlargement is the result of occlusion of the splenic arteries by emboli. It is almost always a sequel of endocarditis of the left side of the heart.

Acute stasis spleen rarely occurs in general venous stasis following diseases of the respiratory or circulatory apparatus, since the intrahepatic branches of the portal vein prevent the occurrence of stasis in the trunk of the portal vein itself. As a rule, acute stasis of the spleen is the result of circulatory obstruction in the portal vein itself, whether the result of hepatic diseases, pylephlebitis, or compression of the portal vein by abdominal tumors, retracting peritonitic cicatrices, etc.

Temporary physiological stasis of the spleen occurs a few hours after meals, because the abundant absorption of the digested masses from the intestines impedes the flow of blood from the splenic vein.

Certain authors claim that the spleen may enlarge in cases of suppressed menses, as the result of arterial congestion.

The most frequent and important form of splenic enlargement is that due to infection. It occurs in infectious diseases. It is rarely absent in intermittent and typhoid fever, and is also observed in typhus and relapsing fever, cholera, yellow fever, dysentery, acute gastro-enteritis, ulcerative endocarditis, acute articular rheumatism, pneumonia, acute miliary tuberculosis, pleurisy, pericarditis, peritonitis, cerebro-spinal meningitis, diphtheria, angina, coryza, variola, scarlatina, measles, erysipelas, pyæmia, septicæmia, puerperal fever, scurvy, splenic fever, glanders, and recent syphilis. Congenital acute enlargement of the spleen is sometimes found in the new-born, if the mother suffered from intermittent fever or syphilis during pregnancy.

In many of these diseases, enlargement of the spleen is not a constant symptom, or at least it cannot always be demonstrated clinically. The severity of the infectious disease does not correspond to the degree of splenic enlargement. The latter not infrequently precedes the other symptoms of the infectious process. In like manner, the enlargement also lasts longer than the other symptoms, because it is the result, not alone of circulatory changes, but of hyperplastic processes in the cells of the spleen. According to Friedreich, there is danger of a relapse in typhoid fever so long as the spleen is enlarged.

It is well known that fine particles of pigment which are introduced into the circulation of an animal are deposited in great part in the spleen and even enter the cells of that organ. As we are justified in regarding bacteria as the cause of infection, it is probable that these likewise find a favorable place of deposit in the spleen. The cells of the spleen will then react with special facility to the irritation produced by the bacteria, because these cells form an intermediate condition, inasmuch as they are awaiting transformation into higher cells (red blood-globules).

II. Anatomical Changes.—The spleen may attain six times its normal dimensions.

In traumatic enlargement of the spleen, evidences of injury are generally found in the neighborhood of the organ.

Embolic enlargement is characterized by the wedge-shaped infarction (vide page 46).

In stasis spleen, there is striking distention with blood, together with changes in the district of the portal vein.

In infection spleen, as a rule, the capsule is transparent and tense, and is only wrinkled when the process is undergoing involution. The splenic pulp is soft and diffluent, but this is partly the result of post-mortem change, since it is found even though the splenic tumor was felt to be quite hard during life. The spleen is often so soft that fine distinctions in its structure cannot be recognized. Occasionally, it contains wedge-shaped foci, whose origin is not clearly recognized.

In the spleen of infectious diseases, the microscope shows congestion, swelling of the splenic cells, increase of the nuclei, fatty degeneration; under certain circumstances, increase of the blood-corpuscle-containing cells, infiltration of the walls of the vessels with round cells, and extravasations of blood.

In sixty-eight cases of sudden death and suicide, Birch-Hirschfeld

found that the average weight of the spleen was five ounces, or 0.26% of the weight of the body.

III. Symptoms.—In the majority of cases, splenic enlargement is only recognized by directing special attention to the organ. Subjective symptoms may be absent, but complaint is sometimes made of tension, a feeling of pressure, or stitches in the splenic region. The pains occasionally radiate even into the left arm and leg. These symptoms may increase in left lateral or even in right lateral decubitus, because the heavy spleen drags upon its ligaments.

Acute enlargement of the spleen is rarely so marked as to be recognizable on inspection. The splenic region would then be prominent, and perhaps, if the abdominal walls are thin and flaccid, a tongue-shaped prominence, which moves with respiration, is seen in the left hypochondrium.

Palpation furnishes the most important results. The spleen can hardly ever be felt unless it is enlarged. If the tumor extends towards the linea alba, indentations can sometimes be felt in its anterior edge. Palpation sometimes gives rise to pain.

During palpation, the patient should assume the right diagonal position, *i. e.*, he lies upon the right shoulder in a position midway between right lateral and dorsal decubitus. The physician stands to the left of the patient, near his head, and the tips of the second, third, and fourth fingers are gently placed in the space between the lowest cortal cartilages and the free end of the eleventh rib. During vigorous respiratory movements the organ will be felt, at each inspiration, projecting beneath the left hypochondrium. If the pressure of the fingers is too great, the spleen is often pushed upwards and backwards, and soft tumors thus escape recognition.

The spleen may be mistaken for digitations of the diaphragm, or the tendinous origin of the left rectus abdominis. In pleurisy, pneumothorax, emphysema, curvature of the spine, and pericarditis, the spleen is sometimes pushed downwards and becomes palpable, although it may not be enlarged.

Splenic enlargement may also be recognized by percussion, although this offers many sources of error. The most frequent source of error is distention of the stomach and colon with firm masses. On the other hand, in meteorism splenic dulness may be absent, despite enlargement of the organ. We must always be on our guard if splenic dulness is enlarged, but the organ cannot be felt. It is suspicious if the increased dulness varies from the ordinary shape of the spleen, if it changes from day to day or after evacuation of the bowels.

In embolic enlargement of the spleen, it is conceivable that a peritonitic friction murmur may be heard and even felt if the infarction has given rise to inflammation of the capsule. Griesinger described intermittent and continuous roaring vascular murmurs in splenic enlargement as the result of intermittent fever during the febrile period. Mosler states that these murmurs occur almost constantly during the period of chill, grow feebler in the hot stage, and disappear in the apyrexial period. Griesinger attributed the murmurs to the large abdominal veins, Mosler to contractions of the splenic artery. Mosler also described a splenic murmur in a case of relapsing fever.

The duration of acute splenic enlargement depends upon the course of the primary disease. In rare cases, the soft spleen ruptures and gives rise to peritonitis and rapid death.

In some cases the enlargement persists and becomes chronic.

IV. Diagnosis and Prognosis.—The diagnosis is not always easy; smaller tumors often remain unrecognized.

The prognosis depends on the primary disease. Rupture of the spleen is so rare that this possibility hardly affects the prognosis.

V. Treatment.—The treatment is almost always included in that of the primary disease. A subcutaneous injection of morphine may be necessary if the pains are very severe. If there is danger that the condition will become chronic, the remedies mentioned in the following section should be employed.

2. *Chronic Enlargement of the Spleen.*

(*Chronic Splenic Tumor.*)

I. Etiology.—This term is applied to those cases in which enlargement of the spleen exists for a long time. It sometimes develops out of an acute enlargement, sometimes it is chronic from the start. In the former event, it is the result of the etiological factors mentioned in the previous section. Some of these causes favor the development of chronic splenic tumor more than others. In acute infectious diseases, for example, acute splenic enlargement is the rule, while causes of stasis, chronic infectious diseases, syphilis, and embolism rather favor the development of chronic splenic enlargement.

In malarial regions, chronic splenic tumors may develop, although there have been no manifest symptoms of intermittent fever. In certain tropical regions, the spleen rarely possesses normal dimensions.

Among the causes of chronic enlargement of the spleen are: leukæmia, pseudoleukæmia, waxy degeneration, neoplasms, tubercle, gumma, and parasites. Chronic splenic enlargement has also been found in rickets and scrofula.

The affection rarely occurs in children and old people. But it is sometimes congenital, and then is often regarded as an indication of hereditary syphilis.

II. Anatomical Changes.—The spleen sometimes attains ten to twenty times its normal dimensions, and it may weigh five to ten kilograms.

The organ sometimes occupies the greater part of the abdominal cavity, compresses and displaces adjacent organs, and extends into the pelvis. The capsule is often thickened, and here and there may contain fibro-cartilaginous deposits. The anterior edge often presents very deep indentations, and these may be more numerous than in the normal spleen. The capsule is often joined to adjacent parts by peritonitic adhesions. If neoplasms or parasites are present in the spleen, they often project above its surface as spherical prominences. Old infarctions are recognized upon the surface of the spleen by slight depressions and a cheesy yellow color.

The transverse section of the spleen varies in appearance according to the character of the lesion. In some cases, there is pure hyperplasia of the splenic tissue, as in leukæmia. In passive congestion of the spleen, the increase of the connective tissue predominates. In other cases, there is a combination of both lesions. In intermittent fever, the spleen is characterized by the abundance of black pigment. In neoplasms and parasites, there is enlargement of the organ, although the splenic tissue proper is not infrequently diminished in amount.

III. Symptoms and Diagnosis.—The remarks made concerning the symptoms and diagnosis of acute splenic enlargement also hold good

with regard to the chronic form. Subjective symptoms may be entirely absent. On palpation, the spleen generally feels firm. As the enlargement is sometimes very great, symptoms of compression appear on the part of the lungs and heart (dyspnœa) or the abdominal organs. According to some authors, chronic ulcers of the leg may be the result of chronic splenic tumor (pressure on the inferior vena cava).

According to Piorry, pressure on the spleen sometimes produces chill and tremor. Naunyn observed cough as the result of percussion or compression of the organ. This symptom disappears after several trials, but reappears after a certain length of time. Swining states that he has observed increased heat in the splenic region. Gerhardt recently described, in a case of aortic insufficiency, a pulsating splenic tumor, and at the same time heard a double sound over the organ. In two cases of acute febrile enlargement of the spleen in patients who suffered from aortic insufficiency, he observed pulsations.

The patients generally have a pale, sallow, sometimes greenish or blackish complexion. They suffer from palpitation or dyspnœa, anæmic sounds are audible in the arteries, and murmurs in the veins, hemorrhages occur into the skin and mucous membranes. They suffer from œdema, and finally die of cachexia. Some of these symptoms, it must be remembered, may be the result of the primary disease.

The disease sometimes lasts for years.

IV. Prognosis.—The prognosis depends on the primary disease. Under specially unfavorable circumstances, the splenic enlargement itself may be the cause of death from the effects of compression.

V. Treatment.—This depends upon the primary disease. If the splenic enlargement is the result of a neoplasm, we can merely maintain the strength of the patient as much as possible by nourishing food and tonics. In gummata of the spleen, astonishing results are often obtained by the administration of potassium iodide. Echinococci of the spleen must be treated surgically. Under more favorable circumstances, internal and external remedies may be employed.

The chief internal remedy is quinine, which may be given by the mouth or, better still, injected subcutaneously (with glycerin and water āā) in the splenic region. If quinine is not tolerated, we may order arsenic (℞ Liq. potass. arsenit., Aq. amygdal. amar., āā ʒ ss. M. D. S., five to ten drops t. i. d. after meals).

If there is pronounced anæmia, we should order iron or iodide of iron, combined with quinine.

Individuals who live in a marshy and malarial region should change their residence.

The various alkaloids of Peruvian bark, piperine, salicin, ergotin, potassium bromide, pilocarpine, etc., have also been recommended to diminish the size of the spleen.

The efficacy of internal remedies may be aided by external measures. According to our own experience, the best plan is the application of ice-bags to the spleen.

The following plans have also been recommended: cold douches to the spleen, faradization, application of iodine, issues or blisters, massage, injections into the spleen of Fowler's solution or carbolic acid. Spontaneous disappearance of the enlargement has been observed after pregnancy.

Extirpation of the spleen (splenotomy) is indicated when death by suffocation threatens, as the result of compression.

3. *Inflammation of the Splenic Capsule.*

Perisplenitis.

I. Etiology.—Perisplenitis is rarely primary (traumatic). As a rule, it is the result of peritonitis or inflammatory processes within the spleen, which have extended to the periphery (generally embolic infarctions, but also all acute and chronic splenic enlargements).

II. Anatomical Changes.—In acute cases, the capsule is covered with fibrinous deposits, which cause the spleen to adhere to surrounding parts, and sometimes form sacs which are filled with pus. In chronic cases, we find fibrous thickenings which have a tendinous appearance, or are thick and almost cartilaginous in hardness. They sometimes constrict the spleen to such an extent as to produce atrophy of the organ. There are often fibrous adhesions to surrounding parts.

III. Symptoms.—The symptoms consist of pain in the region of the spleen, sometimes of a friction murmur. The surface of the organ is sometimes felt to be irregular. The disease is often unrecognized, particularly in acute perisplenitis. Firm thickening may be suspected if the spleen does not enlarge in infectious diseases or portal stasis, and other causes (hemorrhages) have not prevented the enlargement. Fibrous adhesions may be diagnosed if respiratory displacement remains absent, although the spleen can be felt and is not unusually large, or if splenic dulness remains, despite the occurrence of perforation-peritonitis.

IV. Treatment.—Application of ice-bags, otherwise purely symptomatic. If there is severe pain, morphine subcutaneously, warm poultices, cups, sinapisms, blisters, or application of iodine.

4. *Hemorrhagic Infarction and Inflammation of the Spleen.*

Splenitis.

(*Abscess of the Spleen. Splenitis Apostomatosa.*)

I. Etiology.—Splenitis is rarely primary. This is observed as the result of injury (although this gives rise more frequently to rupture of the spleen), and, according to some writers, of great bodily strain. Silberstein reported a case in which abscess of the spleen is said to have resulted from violent sneezing. In many cases the cause cannot be ascertained.

As a rule, splenitis is secondary. It is generally the result of embolism of the splenic artery, which begins with the symptoms of infarction, and generally ends in inflammation. The emboli generally are secondary to valvular diseases of the left side of the heart, more rarely to aneurisms, arterio-sclerotic patches in the aorta, and still more rarely to pulmonary disease.

Changes in the spleen, which are similar to wedge-shaped infarctions, but in which no embolus can be found, occur in infectious diseases (pyæmia, septicæmia, typhoid and relapsing fever, cholera), in protracted, exhausting diseases, and in Bright's disease.

Splenitis is sometimes propagated from adjacent parts. Round ulcer

of the stomach, toxic gastritis, peritonitis, and perinephritis may extend to the spleen, and give rise to secondary inflammation of that organ. Pulmonary gangrene may also extend to the diaphragm and spleen.

II. Anatomical Changes.—Among eighty-four cases of embolism following valvular disease of the heart, the splenic artery was affected thirty-nine times, the renal artery fifty-seven times. The relatively large lumen of the splenic artery, and the slow circulation within it, favor the reception of emboli.

One or more emboli may be present, and occasionally they are so numerous that very little of the splenic tissue remains intact.

A wedge-shaped infarction of the spleen is recognizable by its peculiar shape. The broad base of the wedge is directed towards the surface of the spleen, the narrow apex towards the hilus (corresponding to the distribution of the obstructed artery). In the majority of cases, the infarction extends to the surface. The capsule may then be inflamed, and be covered with peritonitic deposits.

Recent infarctions are blackish-red, granular, and have an hepatized appearance. They gradually undergo decoloration, which begins at the apex and centre, and then extends to the periphery. The color changes to brownish-red, grayish-red, and finally yellow. At the same time the part becomes dry, crumbly, and brittle.

Under favorable circumstances, the infarction may be almost entirely absorbed, leaving merely a depressed, often pigmented cicatrix. In other cases, it undergoes cheesy degeneration and partial calcification. The infarction has a light yellow color, and the relatively well-retained Malpighian bodies appears as pearl-gray dots. Retraction occurs, and the spleen not infrequently becomes irregularly lobulated. Finally, the infarction may terminate in an abscess.

The size of the abscess varies from that of a pea to that of a hen's egg. It sometimes exceeds the dimensions of the infarction and attacks intact splenic tissue. Cases have been reported in which the spleen formed a pus sac surrounded by the capsule, and in which hardly a trace of splenic tissue was left. (Thirty pounds of pus have been discharged from such an abscess.) The spleen sometimes contains numerous abscesses. The pus forms a green, creamy, or light reddish-brown fluid, containing pus-corpuscles, granulo-fatty cells, fat granules, and hæmatoidin crystals. The wall of the abscess is irregular and villous, or smooth and surrounded by a fibrous capsule.

The abscess may perforate into the peritoneal cavity, stomach, transverse colon, pelvis of the kidneys, large blood-vessels, pleura, pericardium, lungs, or externally through the abdominal walls. In very rare cases, the pus becomes cheesy, sometimes calcified in places.

In relapsing fever, splenic abscesses form which are restricted to the Malpighian corpuscles. This has also been observed in typhus fever.

III. Symptoms and Diagnosis.—The diagnosis can only be made under certain favorable conditions. The most important one is the demonstration of a valvular lesion or of other lesions which are known to give rise to embolism. If, under such circumstances, there is a sudden chill (attended in many cases with vomiting), if the patients complain of pain in the spleen, and the area of splenic dulness rapidly increases in size, the diagnosis of wedge-shaped infarction of the spleen may be made.

The diagnosis of splenic abscess is impossible in the majority of cases. In some cases, the lesion is found accidentally at the autopsy, because marked symptoms were absent during life. In other cases, hectic symp-

toms develop: chills, profuse sweats, remittent fever, anorexia, emaciation, diarrhœa, death from exhaustion (phthisis lienalis).

The diagnosis can only be made if fluctuation can be detected in the spleen (which is usually enlarged) and if the affection has been preceded by the causes of splenic abscess. The sudden appearance of masses of pus, coincidently with diminution in the size of the spleen, also arouses the suspicion of splenic abscess. This event indicates perforation into adjacent organs, and the pus may be vomited, expectorated, discharged in the urine or stool, or through the integument. In the latter event, the skin may be undermined, sometimes even to the clavicle or axillary space, before perforation occurs. Perforation into the peritoneal cavity generally proves rapidly fatal after evidences of perforation-peritonitis, but previous peritonitic adhesions may give rise to encapsulation of the pus and prevent free perforation. In some cases, fatal pyæmia sets in because pus and infection-carriers enter the circulation through the splenic vein.

IV. Prognosis and Treatment.—The prognosis of wedge-shaped infarctions is not always unfavorable; that of splenic abscess is grave, and the only hope of a favorable issue lies in judicious surgical measures.

Treatment is purely symptomatic.

5. *Waxy Degeneration of the Spleen.*

I. Etiology.—The causes of waxy degeneration of the liver will also give rise to a similar lesion in the spleen (vide Vol. II., page 213). The spleen is generally the first organ affected, and if death occurs very soon, it may be the only organ which has undergone waxy degeneration. According to Cohnheim, it may develop in four months. In very rare cases, the spleen escapes, while other organs are affected.

Hoffmann found that among eighty cases of waxy degeneration the organs were attacked as follows:

Spleen	74 times	(92.5%).
Kidneys	67 "	(84. %).
Intestines	52 "	(65. %).
Liver	50 "	(62.5%).

II. Anatomical Changes.—Slight grades of the lesion can only be recognized by the aid of the microscope and chemical reagents (vide Vol. II., page 214). Advanced waxy degeneration is recognizable macroscopically, and includes two varieties, viz., the sago spleen and diffuse waxy spleen.

In the sago spleen, the Malpighian corpuscles are affected chiefly or almost exclusively. On sections through the spleen they appear as pearl-gray nodules, which may exceed the size of a pin's head. In some of them, the centre contains a dull-gray dot, which corresponds to the blood-vessel of the follicle. The nodules are often surrounded by a red halo of dilated vessels. On the application of iodine, the degenerated follicles assume a deep mahogany-brown color, which is especially distinct if the waxy follicles are situated within an infarction.

In diffuse waxy spleen, the organ increases in size, and sometimes occupies a large portion of the abdominal cavity. The borders of the spleen

are rounded and blunted, and its consistence is increased. It feels tense, firm, brittle, as if frozen.

On making a section through the organ, large coherent pieces can be scraped off with the knife, and the organ is readily cut into thin sections which are translucent in transmitted light. The organ generally has a flesh-red color, and upon the application of iodine assumes a diffuse, dark-brown color.

Transitional forms between sago spleen and diffuse waxy degeneration are sometimes observed. But we have also seen sago spleen in old cases of waxy degeneration, so that the transition from sago spleen to the diffuse form does not appear to be a necessary event.

The waxy degeneration begins in the capillaries, and then extends to the connective-tissue framework of the spleen. According to Sechtem and Eberth, the splenic cells proper are not affected by the degeneration, but undergo compression atrophy, on account of swelling of the connective-tissue framework. Kyber, Cornil, and others claim that the spleen cells undergo waxy degeneration. Concerning the nature of the amyloid substance, we refer to Vol. II., page 215.

III. Symptoms and Diagnosis.—The condition is not susceptible of diagnosis, unless, after circumstances which usually give rise to waxy degeneration, a hard splenic tumor with round edges is felt, and at the same time there is firm enlargement of the liver and albuminuria; in some cases, diarrhœa points to waxy degeneration of the liver, kidneys, and intestines. Cachexia is often noticed, but it is doubtful whether this depends upon the waxy spleen or upon the primary disease.

IV. Prognosis and Treatment.—The prognosis is unfavorable, although a resolution of the process at the beginning of the disease does not appear to be impossible. The treatment is symptomatic. Special repute is enjoyed by preparations of iodine, iron, and iodide of iron.

6. *Tumors of the Spleen.*

Neoplasms of the spleen possess chiefly an anatomical interest. Some of them, such as fibroma, enchondroma, cysts, dermoid cysts, and cavernoma, are anatomical rarities; others, such as sarcoma and cancer, are more frequent. Cancer of the spleen is generally secondary to cancer of the liver, stomach, or retroperitoneal glands. Six cases of primary cancer have been reported. Medullary cancer is the variety generally observed, and pigment cancer is relatively frequent. Sometimes only a few nodules are found in the uniformly enlarged spleen, sometimes almost the entire organ is destroyed. It may increase very considerably in size, and occupy the larger part of the abdominal cavity. The disease occurs generally beyond the age of 40 years, but occasionally in very young individuals (one case at the age of 12 years). The diagnosis is only possible if cancer is demonstrable in other organs, and the spleen is enlarged, and its surface nodular. The prognosis is unfavorable, and treatment is of no avail.

7. *Parasites of the Spleen.*

Pentastomum denticulatum, cysticercus cellulosæ, and echinococci have been found in the spleen. The two former possess only an anatomical interest.

Echinococcus may occur in the spleen alone, but more frequently it is also present in other organs, generally the liver. We may find either simple sacs, or the latter may contain daughter vesicles. The organ may increase very markedly in size, and compress adjacent organs (lungs, heart, stomach, intestines, or bladder), as shown by dyspnœa, vomiting, constipation, and dysuria. Examination discloses a splenic tumor which moves with respiration, occasionally peritonitic friction sound and prominences. The latter correspond undoubtedly to echinococcus vesicles if they present fluctuation, but the tumors are not infre-

quently hard and firm. An exploratory puncture may be made, but this does not always afford positive results, since the fluid may contain albumin, and is sometimes destitute of echinococcus hooks. The patients often complain of pain in the splenic region. Hectic symptoms set in if the vesicles undergo suppuration. The disease may last more than sixteen years. Recovery can be effected by surgical measures alone. Otherwise death occurs from marasmus or suffocation.

8. *Rupture of the Spleen.*

I. Etiology.—When the organ is healthy, rupture of the spleen may occur as the result of injury in the splenic region. In acute enlargement of the spleen, rupture may take place spontaneously because the capsule is unable to resist the increasing pressure, or it is the result of trifling causes (lifting, vomiting, coughing, etc.). This happens most frequently after typhoid or intermittent fever, but may also occur in typhus, cholera typhoid, and even in miliary tuberculosis.

II. Symptoms and Anatomical Changes.—The accident is generally attended with the symptoms of severe internal hemorrhage. The patients may experience a sensation as if something had burst internally. They complain of pain in the abdomen, confined at first to the spleen; the skin becomes cool and pale, the features sunken; then follow syncope, vomiting, imperceptibility of the pulse, increased splenic dulness, muscular twitchings.

As a rule, death ensues; but a case of recovery has been recently reported. Cicatrization can be looked for only when the rent in the capsule is insignificant. Death may occur at once, or life is prolonged one to two, rarely several days. At the autopsy, the abdomen is found filled with fluid and coagulated blood; peritonitic changes are generally absent. The capsule generally contains a single irregular rent.

Cohnheim described a case in which varicose dilatations of the splenic vessels had ruptured.

III. Diagnosis, Prognosis, Treatment.—The diagnosis is based on the fact that, acute enlargement of the spleen being present, there are sudden signs of internal hemorrhage, pains in the region of the spleen, and increasing dulness. Prognosis unfavorable. Treatment: ice bag, ergotin subcutaneously, opium to relieve the pain, camphor and wine against threatening collapse.

9. *Changes in the Position of the Spleen.*

(*Wandering Spleen.*)

Changes in the position of the spleen may be congenital or acquired, temporary or permanent. Congenital dislocations include the situs viscerum inversus, in which the spleen is situated on the right side, the liver on the left. This condition may be confined to the liver and spleen, or involve the other viscera.

In pleurisy, pneumothorax, spinal curvature, and deformity of the thorax, the spleen is not infrequently displaced downwards; in meteorism, abdominal tumors, and the like, it is often pushed upwards.

The spleen sometimes sinks so far that it is felt below the epigastrium, most frequently in the left iliac fossa, but sometimes in the pelvis or right iliac fossa. It may be so movable as to change its situation in different positions of the body, and may even be turned around its own long axis. In other cases, the mobility of the spleen is impeded by adhesions to adjacent organs. The hilus generally looks upwards, while the anterior edge is situated along the anterior abdominal walls.

The development of this condition is favored by a long, flaccid gastro-splenic ligament, by a blow in the splenic region, lifting heavy loads, and diseases attended with cough. A fruitful source of displacement is the increased weight of tumors of the spleen, which drag upon the ligaments. The gastro-splenic ligament, with the splenic arteries and veins and the pancreas, are drawn into a long cord, which is often twisted upon its long axis. The vessels of the spleen may undergo obliteration or the spleen may be freed from its ligaments, and then undergoes fatty degeneration and atrophy.

The objective symptoms consist of the demonstration of a tumor in the shape of the spleen. In one of my cases, the tumor could not alone be felt through the thin abdominal walls, but the pulsating splenic artery could also be palpated in the hilus. Splenic dulness was absent from its usual position, and did not reappear until the tumor was replaced in the left hypochondrium.

Some patients are entirely free from subjective symptoms; others complain of a feeling of traction and pain; adhesion to the bladder and rectum may produce vesical and rectal tenesmus. Pressure on the nerves sometimes gives rise to formication and paralysis of the lower limbs. In Kiepert's case, pain in the left shoulder was produced whenever the spleen was compressed.

A case has been reported, in which pressure of the dislocated spleen upon the ileum produced death from intestinal occlusion. In another case, coils of the ileum had become incarcerated in an abnormal fissure within the elongated gastro-splenic ligament. Gangrene and dilatation of the stomach have also been observed, the former from excessive stretching and occlusion of the arteries of the fundus, the latter from pressure of the stretched pancreas upon the duodenum.

Bandages may be employed to keep the spleen in place, and as a last resort splenotomy may be performed.

SECTION IX.

DISEASES OF NUTRITION.

1. *Obesity. Polysarcia.*

I. Etiology.—The term obesity is applied to an excessive accumulation of fat in the subcutaneous cellular tissue and in those internal localities (mediastinum, epicardium, omentum, mesentery, appendices epiploicæ, renal capsule, etc.) which are characterized, under healthy conditions, by the presence of a large amount of fat.

The disease not alone produces great annoyance, but also serious danger to life.

The causes are either indirect (predisposing) or direct, but both sets of causes co-operate in the majority of cases.

Heredity is the chief predisposing cause. In some cases, only a few members of a family are affected. In all probability, it is the result of an inherited defective power of oxidation of the cells.

Age is a potent etiological factor. The disease is often observed during infancy and beyond the age of forty years, while childhood and early adult life generally escape. According to some writers, the tendency to obesity is particularly evident in men between the fortieth and fiftieth years, in women beyond the age of fifty.

The female sex presents a greater predisposition to the disease than the male sex.

The more sedentary the habits of life the greater is the danger of obesity; hence it is frequent in those who retire from business, or who are deprived of the use of the limbs on account of amputation or of other causes which act in a similar manner.

Racial differences are also manifested. Thus, obesity is frequent among Hungarians, Wallachians, Orientals, South Sea Islanders, and Hottentots.

A damp, warm climate favors its development; hence the tendency of the Dutch to corpulence.

These factors would probably prove insufficient in many cases, were it not for errors in diet. The latter may be excessive in amount or improperly constituted.

Apart from water and salts, our food consists of albuminoids, fats, and carbo-hydrates. The fat of the tissues is derived mainly from the albuminoids, which are oxidized into nitrogenous and non-nitrogenous substances, the latter representing the fat-producers. Whether the fat contained in the food is converted directly into the fat of the body has not been positively settled, but, at all events, this mode of formation is quantitatively inconsiderable, when compared with the

former method. Fat is not produced from carbo-hydrates unless these are ingested in very large quantities.

The fat derived from the albuminoids of the food is at first destined for further oxidation into carbonic acid and water. Hence, if the ingestion of albuminoids and the corresponding production of fat is excessive, it becomes possible for the oxidizing capacity to be insufficient to decompose the fat, so that it is deposited in excessive amounts in the tissues. In practice, this mode of development of obesity is much less frequent than that arising from the irrational combination of albuminoids and carbo-hydrates.

If, in addition to albuminoids, an excessive amount of carbo-hydrates is ingested, the latter, being more readily oxidized than the fats formed from albuminoids, are first at the disposal of the oxidizing powers of the organism, so that the fats remain unoxidized and are deposited in the tissues.

Hence, obesity is so apt to occur in individuals who are devoted to the pleasures of the table, and, in addition to large quantities of albuminoids, also partake of farinaceous articles, beer, wine, and other alcoholics.

These considerations render it evident that the amount of food ingested should be regulated by the destructive disassimilation going on within the body. If a man, who has been previously active, retires from business, and at the same time continues to take the same amount of food as before, he runs the risk of becoming obese, because the oxidative processes, which are diminished on account of rest, no longer suffice to oxidize the fats which are produced in the body. Diminished oxidation of fats is also, probably, one of the elements in hereditary obesity, since such individuals are often characterized by flabby bodies and phlegmatic temperament, and these are supposed to be associated with diminished oxidation.

Among the immediate causes of obesity are losses of blood, because the diminution in the number of red blood-globules interferes with the power of oxidation. This is also true of anæmic conditions, and, hence, obesity is not uncommon in chlorosis, progressive pernicious anæmia, phthisis, scrofula, and even the first stages of cancer. For similar reasons, it may develop during convalescence from severe diseases.

Attention has often been called to the relations between obesity and disturbances of the sexual organs and functions. Obesity has been observed repeatedly in individuals with imperfectly developed genitals. In men and women, castration; in women, amenorrhœa and sterility are said to give rise to obesity. In many of these cases, however, the cause and effect are mistaken for one another. Women sometimes grow very fat after the first pregnancy, especially if they do not nurse the child.

Cases of congenital obesity have been reported. Wulf described a case in which a still-born infant measured 62.5 cm. in length, and weighed seventeen pounds.

II. Anatomical Changes.—The panniculus adiposus is unusually developed, and in some places, especially in the abdominal walls, forms masses of fat as broad as the hand. The muscles often have a pale, brownish-yellow or sallow yellow color. In very advanced obesity, there is a considerable development of fat in the intermuscular connective tissue, which sometimes results in compression atrophy of the muscular fibres and partial fatty degeneration. The medulla of the bones is often unusually rich in fat.

The mediastinal cellular tissue generally takes part in the abnormal deposit of fat. This is also true of the subepicardial tissue, and results occasionally in compression atrophy and fatty degeration of the muscular fibres of the heart. The left ventricle may undergo hypertrophy. Atheromatous changes upon the intima of the great vessels are found not infrequently. The serum of the blood sometimes has a milky emulsive character (lipæmia), the result of the presence of an unusual amount of fat in the form of granules.

The greater omentum is particularly fatty. In Boerhave's case, it weighed ten pounds. The appendices epiploicæ are converted not infrequently into large clumps of fat. The capsule of the spleen also contains a large amount of adipose tissue, so that deep incisions must be made before the organ is reached. The epithelium cells of the renal tubules sometimes contain accumulations of fat drops. The liver is still more frequently in a condition of fatty degeneration. The diaphragm is pushed upwards on account of the accumulation of the fat in the abdominal organs.

III. Symptoms.—The symptoms develop very gradually, as a rule, but in rare cases they assume a more acute course, the patients becoming visibly larger within a few weeks. The increase in the size of the body appears first and earliest in those parts which, in healthy individuals, contain a well-developed panniculus (cheeks, chin, nipple, nape, shoulders, extensor surface of the limbs, dorsal surface of hands and feet, abdominal walls, mons veneris, labia, and buttocks).

The shape of the body approximates that of a globe. The cheeks are flabby, the palpebral fissure is made smaller by the pushing upwards of the lower lid, the chin appears drawn inwards, because one or more fat folds of the skin project beneath it (so-called double chin). The features appear expressionless, flaccid, almost stupid. An arcus senilis develops prematurely.

The neck is short, the nape contains thick transverse folds, the chest and abdomen are particularly obese, and the abdominal walls often hang down upon the thighs. The umbilicus is either unusually sunken, or very prominent. Umbilical hernia is not uncommon. The genitals often appear buried in the surrounding masses of fat, and varicocele is not an infrequent complication. The buttocks often attain a monstrous size, and the hemorrhoidal veins around the anus are often dilated.

The gait is generally waddling, and, on account of the change in the position of the centre of gravity, the head and upper part of the trunk are held backwards.

The greatest weight hitherto attained is six hundred and nine pounds, although it is claimed that one individual weighed eleven hundred pounds (?).

The following table shows the enormous weights sometimes attained by children.

A boy, æt.	1½ years	53 pounds.
A girl, æt.	4 years	82 "
A girl, æt.	4 years	137 "
A boy, æt.	5 years	189 "
A boy, æt.	11 years	200 "
A girl, æt.	11 years	450 "

The specific gravity of the body diminishes with increasing obesity, so that the individuals readily float in water.

According to the complexion, we distinguish plethoric and anæmic

obesity. Plethoric obese individuals have a red, congested face, and complain of a rush of blood to the head, frequently of dizziness and ringing in the ears; anæmic obese individuals have a pale appearance.

The skin is generally soft and delicate; it exhibits a tendency to eczema intertrigo, particularly in the lower fold of the breast, the umbilicus, and glnteal folds. Acne vulgaris and even acne rosacea are frequently observed.

There is generally increased secretion of sebum and perspiration. The sebum accumulates not infrequently in the folds of the skin, where it decomposes and emits a nauseous odor. The well-known fatty sweat is a combination of perspiration and sebum.

The temperament of the obese is generally phlegmatic. They avoid exercise as much as possible, on account of the discomforts connected with it. Many exhibit an unusual degree of somnolence. There are frequent complaints of rheumatoid muscular pains, probably as the result of the opportunity for catching cold offered by the profuse sweats.

The pulse is often very rapid, and not infrequently more than 100 a minute. Dyspnœa is a common symptom on account of the enfeebled action of the heart, the impaired movements of the lungs (displacement of the diaphragm upwards), and the diminished amount of hæmoglobin in the blood. Œdema and varicose veins in the legs are observed not infrequently as the result of circulatory stasis.

The percussion sound over the thorax is diminished in intensity on account of the thickness of the integument; the respiratory murmur is feeble, partly for the same reason, partly on account of the diminished respiratory movements.

Marked dulness over the sternum indicates a large accumulation of adipose tissue in the mediastinal cellular tissue. Cardiac dulness is sometimes enlarged, and the apex beat displaced externally. The heart sounds are feeble, and systolic murmurs are sometimes heard, or, if the left ventricle is hypertrophied, the second aortic sound is intensified, and may be metallic in cases of arterio-sclerosis. Leichtenstern found a diminished amount of hæmoglobin in the blood. Fatty liver may be suspected rather than demonstrated by physical exploration, on account of the thickness of the abdominal walls (vide Vol. I., page 54 and Vol. II., page 211).

Females often present menstrual anomalies, early cessation of the menses, sterility, catarrh of the genital mucous membrane, uterine displacement, and ovarian diseases. These result from circulatory disturbances, and displacement of the sexual organs by the fatty masses in the abdomen. Sexual desire is often diminished in men and women. In males, Kisch often found absence of spermatozoa in the semen, and this sometimes increased to azoospermia. Sterility in obese individuals may be the result of various causes; they may even be purely mechanical in character, since an abundant development of fat around the genitals may interfere with coitus.

The urine may often contain a sediment of uric acid and urates, more rarely oxalate of lime—the result of an excessive supply of albuminoids in the food and their imperfect oxidation. Sugar is sometimes found temporarily in the urine, and this phenomenon may be a prodrome of a subsequent diabetes mellitus.

Gastric and intestinal catarrh is often seen in the obese. It is the result in part of the excessive ingestion of food, in part of mechanical in-

terference with the gastro-intestinal movements and of circulatory stasis. The latter accounts for the frequent development of hemorrhoids.

These individuals can perform very little muscular work, partly because the muscles are often rendered atrophic by the intermuscular proliferation of fat. There is not infrequently a tendency to syncope, and to nervous and hysterical disturbances. These become especially marked in febrile diseases, which are particularly dangerous to obese patients. Death often results from paralysis of the heart. In addition, the increased bodily temperature is reduced with greater difficulty by baths than in lean individuals. The patients are also very sensitive to venesection. According to some writers, the use of mercurials is also dangerous in obese individuals.

Obesity furnishes a predisposition to other diseases, such as gout, calculi in the urinary and biliary passages, and diabetes mellitus. Some maintain that it is often associated with cancer and multiple furunculosis. Rapidly fatal hemorrhage from the pancreas has been observed with relative frequency in obese individuals.

Death may occur from various causes. It often depends on disturbances of the functions of the heart, and either occurs suddenly from heart failure or gradually after progressive symptoms of stasis. Cerebral hemorrhage is frequent, but is rather the result of the arterio-sclerotic changes which are associated with obesity.

IV. Diagnosis and Prognosis.—The diagnosis is evident at a glance. The prognosis is serious. The condition is attended with numerous dangers, and if the patient is unable to restrain himself and renounce certain of the pleasures of life, he will generally meet an early and distressing death.

V. Treatment.—Certain forms of obesity may subside spontaneously. For example, the obesity of infants, which generally disappears when they use their muscles and begin to walk, and when the diet is changed to one less rich in hydrocarbons.

The obesity following losses of blood, anæmia, and convalescence from severe diseases may also disappear spontaneously.

The methods of treatment adopted for the removal of fat from the body are manifold.

Ebstein has shown that a relatively plentiful supply of fats diminishes obesity, and finally relieves it entirely. The fats not alone satisfy the need for food, but also diminish the desire for fluids, so that the plentiful ingestion of fat implies a diminished ingestion of food. It is necessary, however, to carry out the dietetic rules permanently, in order to prevent the recurrence of the obesity.

Ebstein prescribes the following rules: Three meals may be taken daily: *a. Breakfast*, consisting of a large cup of black tea without milk or sugar, ℥ iij. of wheat bread or toasted rye bread, well buttered (at 6 to 6:30 o'clock in the summer; at 7:30 o'clock in the winter). *b. Dinner*, 2 to 2:30 o'clock: soup (often with the marrow of bone), ℥ iv.–vi. of broiled or stewed meat (fatty) with a fat gravy, a moderate amount of vegetables, especially leguminosa, also the varieties of cabbage, but no potatoes or turnips; salad or stewed fruit without sugar; fresh fruit; two to three glasses of a light white wine; shortly after dinner a large cup of black tea without milk or sugar. *c. Supper* at 7:30 to 8 o'clock; in winter regularly, in the summer occasionally, a cup of black tea without milk or sugar,

an egg, a fat roast, ham, sausage, smoked or fresh fish, ℥ i. of wheat bread, well buttered, now and then some cheese or fresh fruit.

In other words, we may recommend fatty articles, such as butter, fat meat, sauces, and ham, *paté de foie gras*, etc. Hydrocarbons (potatoes, farinaceous articles, cakes, sugar, milk, beer, brandy, champagne) must be avoided as much as possible.

In addition to exact recommendations concerning the amount and character of the dietary, the patients should be enjoined against dressing too warmly, they should live in moderately warm rooms, bathe in cold water, not sleep too long, and exercise a great deal.

Anæmic obese individuals should take iron or a course of treatment at Kissingen, Homburg, or Marienbad.

We can testify from our own experience that, under Ebstein's regimen, the patients rapidly diminish in weight, and feel better mentally and physically. But we sometimes come in contact with individuals who have such an antipathy to fatty articles that the method of treatment described cannot be carried out. In some cases, I have found that the patient's stomach becomes very sensitive, so that the most cautious ingestion of solid food produced vomiting and diarrhœa.

Oertel's plan agrees with Ebstein's in that both recommend the smallest amount of food, but Oertel allows extremely little fat and relatively more hydrocarbons. Oertel also attaches great importance to diminution of the supply of water, and endeavors to deprive the body of fluid by ordering Turkish baths, mountain climbing, or subcutaneous injections of pilocarpine. Drinks and soups should be avoided as much as possible, at all events fluids should not be taken until one and a half hours after meals. Oertel furnishes the following dietary: *Breakfast:* a small cup of coffee with milk and sugar, and ℥ i.–ij. of wheat bread and butter. *Dinner:* ℥ vij. of meat, ℥ ij. green salad, ℥ iij. fresh fruit, but no soup. *Supper:* two eggs, ℥ v. meat, a little caviar, and ℥ v.–vij. of light white wine. At a later period, after the obesity and circulatory disturbances have disappeared, the dinner may be supplemented with fish and farinaceous articles āā ℥ iij., and later even ℥ vi. of white wine; cheese and bread may then be added to the supper. If circulatory disturbances are not present, the amount of fluids need not be restricted so markedly.

The well-known Banting cure has been modified by Vogel as follows: *Breakfast:* coffee without milk and sugar, toast or zwieback without butter. *Second breakfast:* two soft-boiled eggs, lean raw ham or lean meat, a cup of tea or a glass of acid wine. *Dinner:* a plate of weak soup, lean meat (cooked or broiled), a few potatoes, a little bread, green vegetables. *Supper:* bouillon or tea, cold meat, lean ham, soft-boiled eggs, salad, a little bread.

According to the latter method, as much albumen as possible is supplied to the system, but the attempt is made, by the slight supply of fats and hydrocarbons, to force the body to use up its excessive amount of fat. Although the Banting method is effective, it cannot be continued for a long time, and not infrequently gives rise to gastro-intestinal catarrh, palpitation, dizziness, syncopal attacks, insomnia, and other nervous disturbances, even insanity. In several cases, Kisch noticed the development of phthisis. Voit showed recently that the dangers and inconveniences of the Banting system may be avoided by withdrawing the fats and hydrocarbons very gradually at the beginning of the cure,

and gradually restoring them a little, after our immediate object is attained.

Voit furnishes the following tables of the daily amounts ingested by healthy individuals, and in the various cures for obesity:

	ALBUMEN.	FAT.	HYDROCARBONS.
Vigorous laborer, . . .	118	56	500
Well-to-do physician, . .	107	89	362
Ebstein, ...	102	85	47
Oertel, ...	155	25	70
Banting, ...	172	8	81

The number of plans of treatment has not yet been exhausted. Tarnier orders a strict milk diet (six to eight pints daily). We may also mention grape cures, massage, and inhalations of oxygen, inhalations of compressed air, iodine wells, salivation, hunger, or sweat cures.

2. *Gout. Arthritis Uratica.*

I. Etiology.—Gout is a change in the nutritive processes in the body which inclines it to inflammations in the various organs and tissues, and often gives rise to the deposit of urates. The joints are affected most frequently, but by no means constantly; the first joint of the great toe is the one attacked earliest and most constantly.

In the majority of cases, there is an hereditary predisposition; the disease appears in one generation after another, or one or more generations may escape.

According to Hutchinson, the younger children of gouty parents are more apt to suffer than the first born; hereditary transmission is more probable when both parents suffer from the disease, and the gout of the father is more apt to be transmitted than that of the mother.

In the majority of cases, auxiliary factors are necessary to cause an outbreak of the disease.

The most important are dietetic errors—heavy meals, excessive ingestion of albuminoids, wine, beer, and other alcoholics. These conditions may suffice to produce gout without the aid of an hereditary predisposition. Hence, gout is mainly a disease of the wealthy classes.

The danger of the development of gout increases if bodily rest is added to luxurious habits. Whether mental strain favors the development of the disease has not been positively settled.

The previous considerations explain the frequent coincidence of obesity and gout, the former to a certain extent forming a prodrome of the latter.

But although the disease results, in the majority of cases, from high living, in rare instances it follows deprivation and insufficient supply of food.

Toxic gout is a special form of the disease. French writers have shown that gout, particularly renal gout, is not infrequent in workers in lead, and there is no doubt that the introduction of lead into the system changes the nutritive processes in such a manner as to produce a uric-acid diathesis.

A peculiar form of toxic gout is that attributed by some writers to gastro-intestinal catarrh, and to the formation and absorption of abnormal products of

disassimilation (uric acid and lactic acid). Others maintain that the gastro-enteritis in such cases is a manifestation of primary gout. Our own observations have led us to coincide in the latter opinion.

England is the classical territory, so to speak, of gout. Next follow France and Holland, while it is less frequent in Germany, Spain, and Italy. According to Charcot, gout is infrequent in Russia, Sweden, and Norway, although the population is strongly addicted to the use of alcohol. These variations are due to dietetic, rather than to climatic conditions.

Age exercises great influence on the manifest development of gout. It occurs almost always from the age of thirty to forty years. It is extremely rare in childhood, but cases have been observed at the ages of six, ten, and eleven years, respectively.

The large majority of cases occur in males, because they are more exposed to the exciting causes.

The symptoms generally occur in paroxysms, so that the causes of the diathesis must be distinguished from those of the individual attacks.

The gouty paroxysm depends not infrequently on excesses in diet or in Baccho et Venere; or it is preceded by unusual mental excitement. Rheumatic causes seem to exert a certain influence; at least, attacks of gout are most frequent in the autumn and spring. It has been noticed in several cases that individuals who suffered from articular rheumatism, not alone are often attacked by gout, but that the latter is apt to attack those joints which had been the site of rheumatic inflammations. Injuries may at once provoke gouty changes.

II. Symptoms.—As a rule, the symptoms do not occur unexpectedly. The patients generally give a history of hereditary gout, or they have long suffered from the symptoms of increasing obesity. Complaints are generally made of increasing girth of body, dyspnœa, palpitation, vomiting, pyrosis, flatulence, constipation, dizziness, ringing in the ears, etc. The patients have a red face, often suffer from acne rosacea, hemorrhoids, varicocele, etc.

The gouty attack, as a rule, is also preceded by prodomata, and rarely develops suddenly. There is an increase in the gastric symptoms, obstinate constipation, sometimes pain in or hemorrhage from existing hemorrhoids; the urine grows scanty and deposits a brick-red sediment of urates, more rarely it is very profuse and pale. In some individuals, the sexual desire is increased. The patients complain of a feeling of oppression in the chest, palpitation, increased dizziness, and rush of blood to the head. They grow moody, hypochondriacal and irritable, and complain not infrequently of insomnia and excitement. There is often a tired feeling in the limbs, dragging and painful sensations in the muscles, especially of the calf, sometimes transitory pains in certain joints. In certain cases paræsthesiæ, paretic symptoms, tremors, and cramps in the calves make their appearance.

The paroxysm of gout is so much more violent the longer the duration of the prodromata.

The manifest symptoms of gout appear most frequently in the form of acute gouty inflammation of the joints, although some gouty patients remain free for life from any affection of the joints.

It is characteristic of articular gout that, in the majority of cases, the metatarso-phalangeal joint is attacked. The second joint of the great toe or the ankle joint is affected more rarely. As a rule, the other

joints are not attacked until after repeated gouty paroxysms. The ones that are most frequently involved are the fingers, and especially the thumb, but also the knee, hip, shoulder, elbow, and the articulations of the clavicle, vertebræ, maxilla, and costal cartilages.

In the first attack, the great toe is generally alone affected, and even in subsequent seizures the same joint may be repeatedly attacked. The inflammation rarely occurs at the same time in several joints, more frequently one joint becomes involved after the other.

Gouty inflammation generally begins suddenly in the middle of the night, usually from twelve to three o'clock. The patient who went to bed almost entirely free from suffering, is suddenly roused from sleep by excruciating pain in the great toe. The pain is described as boring, compressing, burning, sometimes as a painful sensation of cold. The patients groan aloud, throw themselves about in bed, and cannot tolerate the slightest contact. The skin is hot and dry, the bodily temperature increased, and the pulse is hard and accelerated. The pains generally moderate considerably towards morning. The fever also diminishes and diaphoresis generally appears, the sweat smelling intensely sour in some cases. At the same time the local changes in the joint have become more marked. It is uniformly swollen, the integument covering it is very red, almost erysipelatous, is hot and œdematous ; the surrounding integument not infrequently contains varicose vessels. The œdema sometimes extends over the entire dorsum of the foot, and even to the ankle.

During the day, the patient experiences little pain if the limb is kept quiet, but in the following night the suffering returns. This continues on the average for five to ten days, then the pains subsides, and the attack ceases. The redness and swelling of the joint gradually diminish, a prickling, itching sensation is felt, the epidermis desquamates, and the joint remains a little stiff for a while, but soon regains its former mobility. Many patients feel in better health after the subsidence of the attack. The paroxysms are generally so much shorter the more violent the pains.

We must also refer to those symptoms which indicate the accumulation of urates in the blood. Their increase in the blood may be demonstrated immediately before and during the paroxysm. In healthy individuals, uric acid is absent from the blood, or is present only in traces ; in acute gout its amount may reach 0.25 or 1.75 per cent.

In some cases, the urates in the blood escape through the skin, a white deposit being left upon the integument after evaporation of the perspiration. This is proven to consist of urates by the murexide test, its solubility in alkalies, and the deposit of uric acid crystals on the addition of acids.

If a blister is applied to the skin, the vesicle will be found to contain a large amount of uric acid.

Uric acid is readily demonstrated in the blood, exudations, or transudations by Garrod's thread test. For example, the blood is allowed to coagulate, and four to eight ccm. serum placed in a watch-glass, to which six to twelve drops ordinary acetic acid (30%) are added. A cotton thread, which is not too smooth, is then dipped into the fluid, the whole covered with a glass slide and allowed to stand one or two days at a temperature of 16 to 20° C. Crystals of uric acid are then deposited upon the thread. 0.025 per cent is the minimum amount of uric acid which will deposit crystals (two to three). They are readily recognized under the microscope (vide Fig. 6). The contents of blisters may be directly experimented upon in the manner just described. Atkinson states that he has found an abundance of uric acid in the coating of the tongue.

During the attack, the urine is generally scanty, saturated, acid, and often deposits a brick-red sediment. The specific gravity is increased. Garrod found that the amount of uric acid in the urine diminishes very markedly shortly before the attack, and sometimes merely a trace is left. During the first part of the attack, it remains small, but gradually increases, and after the termination of the seizure may even exceed the normal amount.

Stokvis states that the amount of urea excreted during a paroxysm is diminished to one-third the normal. This author also observed diminution of phosphoric acid, especially of that portion which is combined with the alkaline earths.

Acute gout may end with a single attack. This is particularly true of those individuals who ever after refrain from dietetic errors. Despite such self-restraint, another attack may occur as the result of a blow, fracture or dislocation, or a cold. But the majority of patients soon discard their abstemious habits. Nevertheless, from two to five or more years may elapse before another attack supervenes. In other cases, an attack occurs quite regularly at the end of a year, or in the spring and autumn. As a general thing, they occur so much more frequently the more enfeebled the individual.

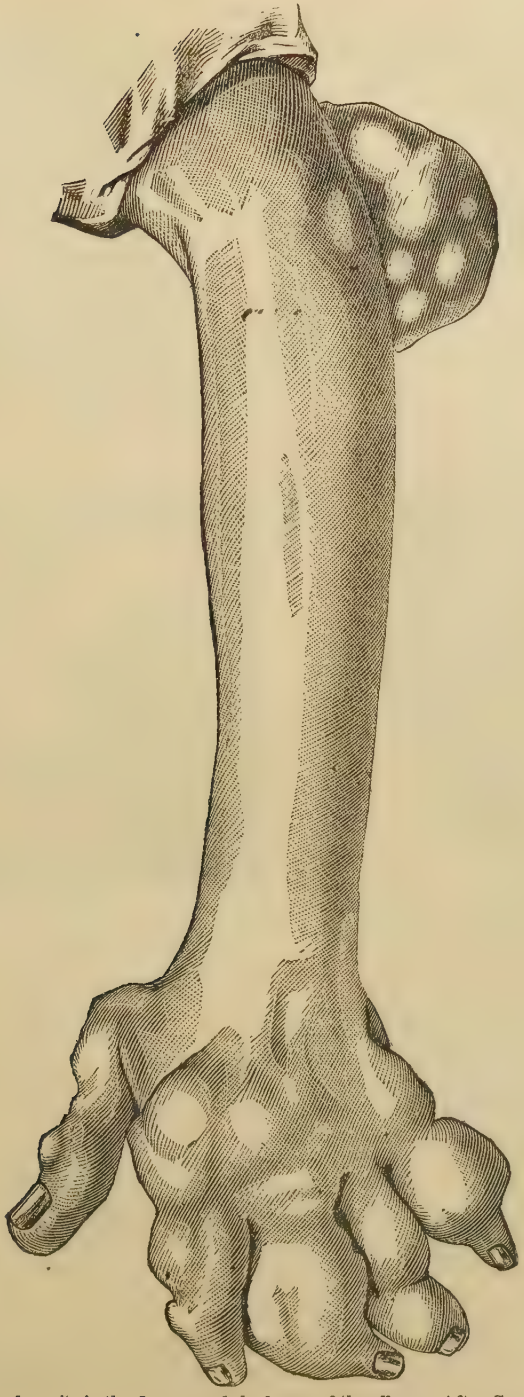
Fig. 6.

Garrod's thread test. Uric acid crystals deposited on a cotton thread. Enlarged 90 times.

The greater the number of the gouty attacks the more they lose their typical character. They become less acute, more protracted, often leave permanent residua in the shape of gouty nodes or tophi; in fine, the symptoms of chronic gout develop.

Chronic gout is often, though not always, a sequel of acute attacks. In rare cases, it appears from the start in a chronic manner. Many joints are generally attacked, particularly those of the feet and hands. As a rule, the joint changes present remissions and exacerbations. The pains in the joints are not so severe as in acute attacks, but the swelling is often greater. The redness of the skin is much less, and often entirely absent. After the pains have disappeared, the swelling subsides very slowly, and does not often disappear entirely. Hard nodes appear in the vicinity of the joint, and increase so much more in size the greater the number of relapses. These nodules are gradually converted into large nodes, which are visible beneath the skin, and feel as hard as a stone. The overlying skin is often pale in the middle, while the periphery is very red and contains dilated vessels. These nodes (tophi) consist chiefly of urates which are deposited upon the outside of the joint. They sometimes attain the size of a cherry, or are even much larger. The overlying skin may inflame and ulcerate, and a chalky or mortar-like mass is discharged, consisting of urates. Under the microscope, this is found to contain fine needles of the urates. These ulcers exhibit very little tendency to heal, and often

Fig. 7.



Gouty deposits in the fingers and the bursa of the elbow. After Garrod.

present luxuriant, easily bleeding granulations. Their base sometimes contains chalk-like tophi, which may assume stalactite shapes.

These tophi may produce such deformity that Sydenham has compared the appearance of the hand to that of a parsnip root (vide Fig. 7). The joints are often movable with difficulty, and may become ankylosed. They often convey a creaking and scratching sensation and sound. Subluxations are produced, so that the terminal phalanges of the fingers are deflected towards the ulnar side, and the other phalanges are bent towards the palm. The entire limb may be rendered useless, and the patient crippled for life.

Chronic gout also attacks the bursæ, fasciæ, tendons, cartilages, bones, and skin.

The bursæ, particularly those of the elbow and patella, become swollen and painful, the skin overlying them is reddened and swollen.

Fig. 8.

Gouty deposits in the cartilage of the ear. After Garrod.

Fig. 9.

Crystals of acid urate of soda, from gouty deposits in the cartilage of the ear. Enlarged 450 times.

After some time, the swelling and pain subside, but firm deposits are discovered which gradually grow larger, and finally form voluminous masses, composed chiefly of urate of soda (vide Fig. 7).

Among the tendons, the extensor tendons of the fingers are affected most frequently, and present more or less hard and stony deposits.

Gout of the cartilages is important in diagnosis. It is observed most frequently in the cartilage of the ear. This contains nodular hard spots which may attain the size of a pea, the centre white, the periphery surrounded by dilated vessels (vide Fig. 8). When pricked with a needle they discharge, on pressure, a white, soft or hard mass, which consists of innumerable fine needles of urate of soda (vide Fig. 9). These nodes are sometimes the only visible evidence of gout. They develop very rapidly (within ten days, in a case reported by Garrod). The overlying skin sometimes ulcerates, and they then fall out of the auricular cartilage. They are not infrequently the site of painful sen-

sations shortly before the onset of an acute gouty affection of the joints.

In rarer cases, gouty deposits occur in the palpebral and nasal cartilages. Virchow has also described gouty deposits beneath the perichondrium of the arytænoid cartilages, and states that these may determine the diagnosis in doubtful cases.

Gout of the skin is rare, but tophi have been observed in the integument of the face. Subperiosteal nodes are observed more frequently.

In chronic gout, the blood is also overloaded with urates, while there is a diminution of the urates in the urine. The phosphoric acid in the urine is also diminished in quantity.

Stokvis calls attention to notable differences in nutrition in gouty and healthy individuals. After the ingestion of inorganic acids (phosphoric or hydrochloric acids), the amount of phosphoric acid in the urine was increased both in gouty and healthy individuals, but in the former there was an increase of the phosphoric acid which was combined with the alkaline earths, in the latter of that combined with the alkalies. After the ingestion of organic acids, the excretion of phosphoric acid was increased in the healthy alone.

Patients who suffer from chronic gout sometimes attain an old age. But in other cases marasmus develops at an early period, asthenic or atonic gout develops, and the patients die from increasing exhaustion. Death also occurs not infrequently from internal or visceral gout (anomalous or latent gout).

There is hardly an organ which may not be the site of gouty organic and functional changes. These may exist independently, or follow previous acute or chronic gout of the joints. In the former event, the diagnosis is extremely difficult, since the symptoms do not differ from those of similar non-gouty conditions. A diagnosis may be made if Garrod's thread test of the blood furnishes positive results. The matter is much simplified if gouty deposits are found on the joints, cartilages, etc. Joint affections, and gouty affections of the internal organs, sometimes alternate rapidly with one another, but the old doctrine of metastasis is not looked upon with special favor at the present time.

Renal gout is the most important form of visceral gout. It produces the symptoms of small contracted kidneys and similar anatomical changes; it is recognizable by the large amount of pale urine of low specific gravity, slight sediment, moderate amount of albumin, and hypertrophy of the left ventricle. It may constitute the sole symptom and terminate fatally.

Not every albuminuria during the course of gout is indicative of gouty kidneys. It may appear as an evidence of cachexia, or as the result of gouty, waxy degeneration of the kidneys.

Cerebral gouty symptoms include headache, hemicrania, syncopal attacks, and epilepsy. Paralysis may occur from encephalorrhagia, the latter resulting from gouty atheromatous changes in the vessels. Psychoses have been attributed to gout in a number of cases. The symptoms of meningitis and myelitis have been ascribed at times to gout of the spinal cord; neuralgiform, paretic, and paralytic symptoms and paræsthesiæ, to gouty changes in the peripheral nerves.

The following ocular changes have been observed: conjunctivitis, with deposits of urates, deposits in the cornea, keratitis, iritis, and affections of the vitreous body. Gouty patients are also said to exhibit a tendency to the formation of cataract. Choroiditis and retinitis are rare.

Impairment of hearing has been observed in some cases, and has been attributed to deposit of urates in the tympanic cavity and the mastoid cells.

The following affections of the circulatory organs are often observed: palpitation, stenocardia, hypertrophy of the heart, myocarditis, dilatation of the heart, sometimes symptoms of stasis, and valvular lesions. Pericarditis may also be associated with gout. The disease also predisposes to arterio-sclerotic changes, and thus to aneurismal dilatations.

Vérité has described gouty rhinitis, which was characterized by the formation of mucous concretions. Catarrh of the air passages, and inflammations of the pleura and lungs may also depend on the gouty diathesis. The pneumonia sometimes terminates in gangrene. Huchard lays stress on the frequent occurrence of gouty hæmoptysis, particularly of the nocturnal variety.

In some cases, functional disturbances ensue in the shape of gouty asthma.

Gouty parotitis, angina, and œsophagismus have been observed. Dyspeptic and cardialgic attacks, vomiting, hæmatemesis, and incontinence of the pylorus have also been described. Functional and ulcerative changes in the intestines occur occasionally.

Gout, *per se*, is perhaps capable of giving rise to cirrhosis of the liver.

Pyelitis, cystitis, or spasm of the bladder may set in, and even mucopurulent discharges from the urethra (gouty gonorrhœa). Inflammations of the testicles and prostate, hydrocele, and indurations in the penis are attributed in some cases to gout.

The skin presents a tendency to inflammations, so that eczema, the various forms of acne, and other inflammatory changes are not infrequent. These are generally characterized by a tendency to relapses and by marked obstinacy. This category also includes the predisposition to phlebectasiæ. In not a few cases, injury to the skin is followed, in gouty individuals, by severe inflammations, accompanied by high fever, and even terminating in gangrene.

Visceral gout often develops in a slow and insidious manner, but it is more dangerous than the articular variety.

Gout is related not infrequently to various other diseases. It often occurs in the obese because both conditions are owing to similar causes. There is no doubt that gouty individuals often develop diabetes mellitus, and that diabetics may suffer from gouty symptoms. Gouty individuals also exhibit a tendency to the formation of calculi in the urinary passages; more rarely in the biliary canals.

In some cases, the patients experience only a single attack of gout in the joints; in others, they recur after intermissions of years, or the patients die after prolonged symptoms of chronic gout, or death is the result of visceral gout, and may occur very rapidly in apparent good health.

III. Anatomical Changes.—The specific changes in gout consist of deposits of urates in various organs. The inflammatory conditions which may be present hardly differ at all from those found in non-gouty diseases.

Gouty joints often contain deposits of urates upon the surface of the articular cartilages. They form whitish, chalk-like masses, which begin as punctate spots, gradually increase in size and height; at first they are sometimes evident only on microscopical examination. The first and most extensive changes appear in the centre of the cartilage, the peri-

pheral parts often remaining intact for a long time, though in many cases their blood-vessels are congested. The more the earthy deposits increase the more the surface of the cartilage is perforated and the substance destroyed and defibrillated. The urates extend occasionally into the adjacent osseous substance.

In recent cases, the synovial membrane shows congestion and loosening of its tissues; in older ones, thickenings, villous proliferations, and earthy deposits.

Gouty concretions are especially apt to form on the outer surface of the capsule of the joint, where they appear as tophi. Garrod observed one in the hand weighing two ounces.

Gouty deposits are also found in the bursæ, tendons, fasciæ, and beneath the periosteum.

Virchow recently described uratic deposits in the spongy portion of the phalanges. Garrod mentions a tendency of the bones to brittleness, as a result of the formation of cavities filled with masses of fat. Gouty deposits have also been observed in the medulla of the bones.

The muscles are sometimes atrophied, especially when the limbs have been inactive for some time.

In the heart, we often find dilatation and hypertrophy, callosities, and fatty degeneration. Chronic inflammation of the endocardium is not infrequent, and these foci of inflammation may contain uratic deposits. Pericarditic changes may also be connected with gout.

Garrod found uric acid in exudations.

The aorta is often the seat of arterio-sclerotic changes, in which Bramson found deposits of urates. Dilatation and aneurism of the aorta may also develop.

Schroeder van der Kolk found urates in the walls of the veins.

The air passages and lungs may exhibit the signs of inflammation; in the lungs, these terminate occasionally in abscess and gangrene. The statements concerning gouty deposits in the lungs require confirmation.

Swelling, inflammation, and even ulcerative destruction of the mucous membrane have been observed in the gastro-intestinal tract.

The liver is often enlarged and in a condition of fatty degeneration or cirrhosis; in the latter event, the spleen may be swollen.

The renal changes appear at times as simple, contracted kidneys (atrophy of the organ, nodular surface, adhesions of the latter to the capsule, atrophy of the cortex, thickening of the walls of the arteries), or urates (occasionally carbonate of lime) are deposited in the form of infarctions in the tubules of the retracted kidneys, or there are interstitial gouty deposits, or a combination of interstitial and intratubular uratic deposits. The former are especially numerous in the pyramids, rarer in the cortex. They sometimes form grayish-white streaks along the straight tubes, or appear as dots at the apices of the pyramids.

Gouty deposits and inflammatory changes are found occasionally in the pelvis of the kidneys, and in the bladder. In one case, Garrod found similar changes in the penis.

In a few cases, deposits of urates have been noticed upon the cerebral meninges, the spinal dura mater, and in the neurilemma.

According to Ebstein, the gouty deposits begin interstitially in all organs. At first, a circumscribed necrotic focus forms in the tissues. After the necrosis is completely developed, the affected part becomes the site of a deposit of needle-

shaped crystals of the acid urate of soda. These often fill the necrotic focus so completely that the latter becomes visible only after the crystals are dissolved; in other cases, the transparent crystalline masses are found surrounded by a bright zone. Finally, as the result of reactive inflammation, the necrotic focus becomes surrounded by a zone of round cells.

The gouty deposits are composed, in great part, of the acid urate of soda. The following tables give the results of chemical examination by two observers:

	Marchand. (Tophi from thigh.)	Lehmann. (Tophi from metacarpus.)
Urate of soda,	34.20	52.12
Urate of lime,	2.12	1.25
Carbonate of ammonia,	7.86	———
Phosphate of lime,	———	4.32
Sodium chloride,	14.12	9.84
Animal matters,	32.53	28.49
Water,	6.80	3.98
Losses,	2.27	
	100.00	100.00

Budd states that he has also detected hippuric acid in the deposits.

Marchand and Lehmann also analyzed the osseous tissues, and found a dearth of earthy matters, and excess of fat.

	Marchand.		Lehmann.		
	Ulna.	Femur.	1st Case.	2d Case.	3d Case.
Phosphate of lime,	43.18	42.12	35.16	35.83	37.22
Carbonate of lime,	8.50	8.24	8.41	9.82	8.99
Phosphate of magnesia,	0.99	1.01	1.31	1.05	1.13
Cartilage,	45.96	46.32	38.14	38.28	40.03
Fat,			12.11	13.37	9.15
Soluble salts,	1.37	2.27	2.93	2.03	1.82

All are agreed that the excess of urates in the blood is the main cause of the production of gouty symptoms, but opinions differ as to the source of origin of the uric acid. This is owing to the fact that we know so little concerning the site of production of uric acid under normal conditions.

As a matter of course, the excess of uric acid in the blood will become still greater if the formation of uric acid is increased by an excessive supply of food, or if, under the influence of lead poisoning or deprivation and want, the processes of oxidation are rendered less active, and a portion of the albuminoids are converted, not into urea, but only into uric acid.

Garrod believes that the paroxysmal character of gout is owing to the fact that the kidneys at times prove unable to excrete the excess of uric acid, so that its transitory accumulation provokes a paroxysm. This theory is opposed by the fact that attacks of gout may occur, although the kidneys are intact. It is, therefore, much more plausible to assume an intermittent increase in the formation of uric acid, rather than disturbed excretion.

The paths of conduction for the uric acid from the tissues to the lymph and blood currents often suffice, but local stasis is apt to ensue if the circulation is disturbed by inflammation, injury, etc. For example, Charcot observed, in a case of hemiplegia, that gout developed only in the joints upon the paralyzed side. The unusually frequent localization of gout in the joints appears to be owing to the fact that the muscular tissue and medulla of the bones take an active part in the production of uric acid which they transmit in part through the lymph channels of the cartilage, and also to the slowness of the circulation in the bones and the consequent opportunity for stasis.

The frequent implication of the great toe is owing perhaps to its extreme peripheral situation, and to the fact that it carries the weight of the body, and is especially subjected to mechanical irritation.

IV. Diagnosis.—Acute gout of the joints is generally recognized very readily from the typical situation and specific course of the disease, assocated with hereditary and constitutional factors.

As a rule, the diagnosis of chronic gout of the joints is also easily made. It has generally been preceded by typical acute attacks, and tophi

are found in the joints, often in the bursæ, tendons, cartilages of the ear and nose, and sometimes of the larynx. It might possibly be mistaken for arthritis deformans, but this could be excluded by examination of the blood and the chemical constitution of the contents of a vesicle (produced by application of emplast. cantharidis).

The recognition of visceral gout may be difficult so long as gouty deposits are not observed. Careful attention must then be paid to the previous history and constitution of the patient, and Garrod's thread test should be made.

V. Prognosis.—The prognosis is always serious. It is doubtful whether the gouty constitution can be subdued permanently, although some patients who are careful in their habits experience only a single attack.

The paroxyms of acute gout of the joints almost always terminate in speedy recovery. Chronic gout often gives rise to deformity of the joints and renders the limbs useless. Visceral gout is always attended with danger, and is sometimes followed quickly by a fatal termination.

VI. Treatment.—The prophylactic measures are the same as those described under the head of obesity.

In an attack of acute gout of the joints, the limb should be kept elevated, in order to favor the outflow of blood, and should be wrapped in salicylated wadding. Schroeder recently recommended inunction of the affected joints with green soap. The patient is placed on fluid diet, and is allowed to drink lemonade. In several cases I think the attack has been shortened by the administration of salicylic acid (gr. vij. every hour) until tinnitus aurium was produced, but the effect is never so pronounced as in acute articular rheumatism.

After the pains have subsideded, the stiffness of the joint may often be relieved rapidly by cautious massage and movement of the limb.

As a general thing, the treatment should not be too energetic, since grave and even fatal symptoms of internal gout have been known to develop under such circumstances. For this reason we should avoid the application of ice, leeches, and blisters to the joint, and the administration of emetics, drastics, large doses of colchicum, etc., to prevent a threatening attack. The warm-water cure of Cadet de Vaux (200-250 ccm. of hot water taken every quarter of an hour, for twelve hours) should not be employed, since this plan is sometimes followed by sudden death.

Acute exacerbations in the course of chronic gout must be treated like the paroxyms of acute gout. Much cannot be expected from internal remedies. Those recommended are: tincture or wine of colchicum (gtt. x.–xij. t. i. d.), aconite, lithium carbonate (gr. iss. t. i. d.), potassium iodide (ʒ ij. : ℥ v., one tablespoonful t. i. d.), and salicylic acid. The functions of the skin should be kept active by lukewarm baths and frictions, and the occurrence of stiffness of the joints should be prevented by massage.

Importance is attached to the use of lithia wells and artificial lithia waters.

Obese patients should take a cure at Marienbad or Carlsbad; if there is a tendency to the formation of uric-acid gravel, at Vichy, Neuenahr, or Ems. Feeble, exhausted individuals may resort to the acratothermal springs (Gastein, Wildbad, Ragaz, Teplitz), while pronounced gouty deposits, gouty ulcers, or cutaneous eruption are treated with sulphur baths (Neundorf, Eilsen, Meinberg, Aix, etc.).

Symptoms of visceral gout generally require stimulating measures,

and also treatment like that pursued when similar symptoms are the result of non-gouty causes. It is also recommended that the gout be diverted from the internal organs to the joints by the application to the latter of blisters, mustard poultices, or other derivatives.

3. *Diabetes Mellitus.*

I. Etiology.—Diabetes depends upon an anomaly of nutrition which is manifested by the permanent excretion of sugar in the urine. It is an independent disease which must be distinguished from transitory, symptomatic excretion of sugar (glyclosuria or mellituria). The disease appears to be increasing in frequency, perhaps because the urine is now examined more carefully than heretofore, perhaps in part as the result of the mental and bodily excesses connected with modern civilization.

Heredity is a prominent etiological factor. Diabetes is sometimes observed in successive generations, sometimes certain generations escape. It is also observed at times in families in which nervous diseases and psychopathies are hereditary. Hereditary obesity and gout also create a predisposition to the development of diabetes.

In certain cases it is the direct effect of a nervous affection, particularly of hemorrhage, softening, and tumors of the floor of the fourth ventricle. Claude Bernard showed that the lesion of a definite part of the floor of the fourth ventricle, near the origin of the pneumogastric, is followed by the excretion of sugar in the urine. Weichselbaum recently observed diabetes in a case of multiple cerebro-spinal sclerosis and, on autopsy, a patch of sclerosis was found in the floor of the fourth ventricle in the part referred to.

Functional nervous diseases, for example, chorea, epilepsy, and psychopathies, may also be associated with diabetes. There is no doubt that it is sometimes the result of strong mental excitement.

The disease is sometimes attributed to injuries, particularly those which give rise to general concussion of the nervous system, also to blows in the region of the stomach, liver, or kidneys.

There can be no doubt that certain cases are produced by cold and exposure to wet, but the circumstances of the case should be carefully examined before it is attributed to such causes.

Improper diet is sometimes a causative agent. Cantani attributes the frequent occurrence of the disease in Italy to the fondness of Italians for farinaceous and sweet food. Injurious effects have also been attributed to abundant ingestion of sugar and carbo-hydrates, or of fruit, new beer and wine.

Some authors claim that diabetes is often associated with sexual excesses.

It develops occasionally after infectious diseases, with relative frequency after malaria, more rarely after typhoid fever, measles, scarlatina, or dysentery. Syphilis, either with or without lesions of the central nervous system, is an occasional cause of diabetes. It has been observed in a number of cases after cirrhosis and abscess of the liver, and portal thrombosis. A similar influence has been attributed to gastric and intestinal affections, and to acute or chronic diseases of the pancreas.

In not a few cases no cause can be discovered.

The disease is more frequent in males, except in children, in whom the females predominate.

It develops most commonly between the ages of 20 to 50 years, a little earlier in females than in males.

A case has been reported in a girl æt. 7 months. Rollo reported one in a man of 77 years.

It is generally assumed that the disease is more common in the well-to-do classes.

II. Symptoms.—The specific symptoms of diabetes are preceded not infrequently by prodromata, consisting of gastric disturbances (changes in appetite, eructations, vomiting, flatulence, irregularity of the bowels) with mental depression, hypochondriacal ideas, dizziness, rush of blood to the head, etc.

In other cases, there are no prodomata, but insatiable hunger and thirst with progressive emaciation rouse a suspicion of the disease even in the patient himself.

Some individuals develop obstinate neuralgias and rheumatoid muscular pains, which are associated with latent diabetes. Bilateral neuralgia is especially suspicious.

Violent pruritus, obstinate eczema, chronic furunculosis and, in women, pruritus vaginæ should lead us to examine the urine for sugar.

Diminution of sexual potency is sometimes associated with diabetes. Nocturnal enuresis in childhood should arouse our suspicion. Complaints of a feeling of dryness in the mouth and throat must also attract attention.

The existence of diabetes is sometimes indicated by certain accidents, such as a peculiar sour, apple-like, or chloroform-like odor from the mouth or urine, white patches on the clothing in places on which drops of urine have fallen (crystallized sugar), or masses of crystals in the night vessel.

In some cases, the ophthalmologist is first consulted, on account of cataract, retinitis, neuroretinitis, ocular paralyses, disturbances of refraction and accommodation, etc.

In two instances I observed the occurrence of death during apparent good health. The patients suddenly lost consciousness, breathed stertorously, and died in coma. In both cases the bladder was found very markedly distended, and the urine contained sugar. The patients were females, æt. 20 and 24 years respectively.

Obesity, gout, and calculi are associated not infrequently with diabetes and should lead to an examination of the urine.

The quantity of urine is almost always increased, and may amount to 3,000 to 10,000 ccm. or even more (normally 1,500 to 2,000 ccm.). Biermer described a case in which 16 litres, Harnack one in which 18½ litres were passed daily. In many cases there is, at first, an excessive secretion of urine, but the latter does not contain sugar. In other words, the diabetes mellitus is preceded by diabetes insipidus. The reverse condition also obtains in some cases. The patients pass water frequently and are often disturbed at night. According to Lecorché, a larger amount of urine is passed at night in the early stages, during the day in the later stages.

In some cases the amount of urine voided never exceeds the normal amount.

The urine is unusually pale and sometimes is hardly distinguishable

from water; it is so much lighter in color the larger the amount. The urine is generally clear, froths readily and for a long time, and rarely contains a sediment.

In some cases the excretion of fat in the urine has been described. In a recent case, the excretion of fat and sugar was intermittent, and disappeared when the patient was placed on low diet.

The urine generally possesses a peculiar sickening odor, occasionally it resembles the smell of fruit or chloroform. In the latter event, the urine usually turns a dark cherry red on the addition of dilute chloride of iron and often contains acetone. The urine has a more or less sweet taste.

On account of the insatiable thirst, children sometimes drink their own urine.

Its reaction is almost always acid and remains so even after it is exposed to the air for a long time. This is owing to the fact that lactic acid is one of the products of fermentation of sugar. The specific gravity is increased in almost every case. It ranges from 1.030 to 1.050, or more (normally 1.015 to 1.020). It may be laid down as a rule of practical importance that a large amount of urine of a normal or increased specific gravity is indicative of diabetes.

In rare cases, diabetic urine has an exceptionally low specific gravity (1.008 to 1.002).

It has been claimed by some authorities that sugar is present in normal urine. But, at the most, normal urine contains merely traces of sugar, which cannot be discovered by the ordinary methods of examination.

The average daily amount of sugar excreted is six to ten ounces, but it may reach one to two kilograms or more. The percentage of sugar may reach ten to fifteen per cent.

The sugar may vary considerably in different portions of urine, so that, in making quantitative tests, it is well to take the entire quantity passed in twenty-four hours. It increases in amount the greater the quantity of sugar and starch ingested. If the patients are placed on an exclusively nitrogenous diet, the sugar in the urine disappears entirely in many cases, in others it persists in smaller amounts. The former cases are the milder, the latter the graver ones from a prognostic standpoint. The latter generally develop out of the former, so that we are justified in speaking of a mild first stage and a severe second stage of the disease. Kuelz found that there are mixed forms, *i. e.*, the same patient will sometimes present the symptoms of the first stage, at other times those of the second stage. While fasting, the excretion of sugar ceases in many cases, but not in all. As a rule, muscular effort diminishes the excretion of sugar, in rarer cases it increases it. During febrile diseases, the sugar sometimes diminishes and even disappears, while mental excitement not infrequently produces marked increase.

During the first stage of diabetes mellitus, sugar is sometimes entirely absent from certain portions of the urine. Even at a later period, there are sometimes marked differences between the daily and nocturnal urine, and the latter may contain merely a trace of sugar. If the diagnosis is

doubtful, the patient should be allowed to partake freely of saccharine and farinaceous food, and the urine examined two to four hours later. As a rule, the excretion of sugar dependent on the food ceases at the end of six hours.

Before the examination for sugar is made, the physical qualities of the urine (quantity, color, reaction, specific gravity, odor) should be known. If the urine contains albumin, it should be boiled in a test-tube and a few drops of dilute acetic acid added to remove the albumin. The tests for sugar are then applied to the filtered urine.

Moreover, if the urine is not clear, it must first be filtered before the tests for sugar are employed.

The most convenient practical test is Moore's or Heller's test. One fifth of the test-tube is filled with urine, about one-third caustic potash is added, and the upper layers are heated over a spirit lamp. The heated parts grow yellow, then reddish, finally brownish-red and mahogony colored. The light yellow color is produced often in normal urine; the mahogony color alone is decisive to a certain extent. If a drop of nitric acid is added to the boiling urine, it bubbles over and emits the odor of burnt sugar or molasses.

If positive results are obtained, Trommer's test should be applied, after the manner modified by Salkowski. A test-tube is filled one-fifth full with urine, and about a third part of officinal liquor potassæ is added. A ten-per-cent solution of sulphate of copper is then added, drop by drop. With each drop is formed a light blue, cloudy deposit of hydrated oxide of copper, which at first dissolves completely on shaking, and gives a beautiful deep blue color. The drops of acid are added until the hydrated oxide of copper no longer dissolves completely. The upper layers are then heated to boiling, and if sugar is present they will turn yellowish-red from the production of suboxide of copper. This color will spread through the fluid even if the tube is removed from the flame. A deposit of the suboxide, after prolonged boiling or on cooling of the boiled urine, is not proof of the presence of sugar.

If the tests are not decisive, the urine may be filtered through animal charcoal, then diluted with two to four times the amount of water, and Trommer's test employed.

Other tests may also be tried. The following is that suggested by Boettger: the urine in a test-tube is mixed with an equal volume of a solution of sodium carbonate (1:3) and a little basic nitrate of bismuth added. After prolonged boiling, metallic bismuth will be deposited, if sugar is present, as a gray, then a black precipitate.

In doubtful cases, the fermentation test may be employed. In the presence of yeast, grape sugar undergoes alcoholic fermentation, and is split into carbonic acid and alcohol, with glycerin and succinic acid as by-products:

$$\underset{\text{(grape sugar)}}{C_6H_{12}O_6} = \underset{\text{(æthyl alcohol)}}{2C_2H_6O_4} + \underset{\text{(carbonic acid)}}{2CO_2}$$

Into a fermentation tube (vide Fig. 10) urine is poured until the long vertical arm of the tube is filled with urine. Shaking the tube and the formation of froth must be avoided, since, after sufficient urine has been added, it should come directly in contact with the top of the long arm. A piece of compressed yeast as large as a pea is then added, and finally the long arm is separated from the bulb by the introduction of mercury. If the apparatus is put in a place which is not too warm (not over thirty degrees), bubbles of carbonic acid begin to form within a few hours, and rise to the top of the straight tube. The test is only decisive when we are sure that the yeast is free from sugar, and to determine this point a similar experiment should be made with pure water.

A somewhat more complicated fermentation apparatus is shown in Fig. 11. Into B are placed thirty to fifty ccm. urine, to which yeast is added, and lime or baryta water is placed in B′. The carbonic acid developed in B′ passes through the tube t' to B′, where it produces carbonate of lime or barium, as shown by increasing cloudiness.

The quantitative analysis of sugar in the urine may be made with Fehling's solution or the fermentation test, but we must refer the reader for details to the treatises on urinary analysis.

It is now generally believed that grape sugar is not the only form of sugar in diabetic urine.

In a few cases it contains inosit, which does not affect the plane of polarization, and does not ferment or reduce an alkaline solution of oxide of copper. Vohl reported a case in which the grape sugar was gradually replaced by inosit, so that the diabetes was converted into pure inosituria.

In one case, Reichardt found dextrin in the diabetic urine.

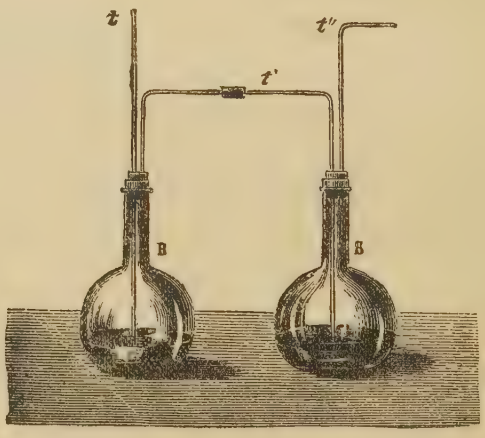
The urine may also contain fruit sugar (levulose). This deflects the plane of polarization to the left, and reduces the salts of copper.

But Worm-Mueller has shown that the urine contains substances which are not varieties of sugar, but nevertheless possess the property of deflecting the

Fig. 10.

Fermentation tube, one-fourth natural size.

Fig. 11.



Glass bottle apparatus for fermentation test.

plane of polarization to the left; for example oxybutyric acid. Hence, the presence of levulose is not proven unless the supposed substance is capable of undergoing fermentation.

Great importance has been attached to the so-called iron-chloride reaction, and to the demonstration of acetone in the diabetic urine. If ferric chloride is diluted until it assumes the color of Rhine wine, and then added drop by drop to a test-tube half filled with urine, a dark cherry-red or Burgundy color (so-called ferric chloride reaction) appears not infrequently in diabetic urine, in addition to the flocculent cloudiness produced by the deposit of phosphates. It should be distinguished from the reaction observed on adding ferric chloride to the urine of diabetics who have taken salicylic acid. In the latter event, the color is a violet blue, and is not transparent.

Diabetic urine which gives the ferric chloride reaction generally has a peculiar sour, aromatic odor, somewhat like that of chloroform or apples, and acetone (and alcohol) has been discovered in the urine in such cases. It was very natural that a connection was sought between the ferric chloride reaction and the acetone in the urine. This was supposed to be proven by Gerhardt and Geuther's case, according to which the ferric chloride reaction is produced by æthyldiacetic acid, and which, under the action of alkalies and after the absorption of water, is decomposed into acetone, alcohol, and carbonic acid:

$C_6H_{10}O_3$	+	H_2O	=	C_3H_6O	+	C_2H_6O	+	CO_2
æthyldiacetic acid		water		acetone		æthyl alcohol		carbonic acid

According to this view, the ferric chloride reaction is owing to the presence of æthyldiacetic acid in the urine, and the apple odor to one of its products of decomposition, viz., acetone. Since diabetics who presented the reaction in question sometimes diffused the odor of acetone in the expired air, and Petters discovered acetone in the expired air of diabetics, there was a tendency to attribute to acetonæmia (overloading of the blood with acetone) those conditions of coma and grave impairment of the central nervous system (diabetic coma) in which the patients not infrequently die more or less suddenly. In recent times, however, this theory has met with a good deal of opposition.

In the first place, Fleischer showed that the chloride of iron reaction sometimes appears in diabetic urines which do not emit the odor of acetone, and also in others which do not contain æthyldiacetic acid. Hence it appears that this reaction, perhaps even in the majority of cases, is not connected with æthyldiacetic acid. Furthermore, Deichmueller and Tollens found acetone but no alcohol in one specimen of diabetic urine, thus controverting the opinion that acetone is produced by the decomposition of æthyldiacetic acid. It appears probable, therefore, that in many cases the ferric chloride reaction is the result of an abnormal fermentation of the sugar, for which Markofnikoff and Fleischer assume a special acetone ferment.

With regard to acetonæmia, it must also be remembered that human beings can tolerate large doses of acetone, and the view is gaining ground that diabetic coma is the result of various conditions.

The ferric chloride reaction and acetone in the urine are not alone not constant in diabetes mellitus, but have also been observed in infectious diseases, febrile conditions in general, and cachexia.

The ferric chloride reaction sometimes appears in diabetes mellitus or becomes very marked when the patients are placed on a strictly meat diet. Ebstein observed it increase on the occurrence of typhoid fever. It sometimes continues for days, and gradually subsides. If the urine is allowed to stand for some time, the reaction gradually grows weaker.

In addition to sugar, acetone, and alcohol, diabetic urine sometimes contains albumin. Slight albuminuria (often transitory) is not unusual. It may be the result of cachexia, calculi in the urinary passages, cystitis, perhaps of an implication of that part of the floor of the fourth ventricle whose irritation, according to Bernard, produces albuminuria. True nephritis is rare. Frerichs observed it sixteen times among three hundred and sixteen cases of diabetes. In one of my patients who returned from Carlsbad with the urine free from sugar, severe parenchymatous

nephritis developed at the end of a month, and proved fatal in three weeks.

The daily amount of urea in the urine is increased. Examinations in healthy and diabetic individuals, living under the same external conditions, and with the same diet, have shown that the latter produce more urea, *i. e.*, decompose more albumin. The daily amount excreted sometimes exceeds five ounces. The amount of urea and sugar are not directly connected with one another, and although both increase and diminish simultaneously, nevertheless there are numerous exceptions to this rule.

The amount of uric acid is diminished; occasionally, a sediment of urates is deposited. Budd states that in diabetics who suffer from renal gravel and calculi, the symptoms referable thereto grow less with the increasing severity of the diabetes.

The kreatinin may be slightly increased or diminished in amount. If fever occurs, the urea, uric acid, and kreatinin increase as in non-diabetics.

Wickl states that the amount of hippuric acid is increased.

Hallevorden and Leube noticed increased excretion of ammonia.

While the amount of chlorides is unchanged, that of the phosphates and sulphates is increased. The relation between the phosphates and urates varies. Teissier observed cases in which all the symptoms of diabetes mellitus (increased thirst, hunger, increased urine, furunculosis) were present, with the exception of sugar in the urine, while the phosphates were considerably increased; in other cases, the presence of sugar alternated with increased excretion of phosphates (phosphatic diabetes). In one case, Fuerbringer noticed the alternation of sugar and oxalic acid in the urine.

The excretion of lime and alkalies in the urine is increased in diabetes.

Urinary sediment is generally absent, but urates and oxalate of lime have been observed in a few cases. Renal casts are sometimes present in nephritic complications. Leptothrix has been observed in freshly passed urine. If the urine is exposed to the air and undergoes fermentation, it contains cloudy opacities which are composed of yeast cells.

Insatiable hunger and unquenchable thirst are among the most frequent symptoms of diabetes. In three of Petters' cases, five to eight litres fluid were drunk daily, and Dupuytren reports that one of his patients ate in a single day an amount of meat which almost equalled one-third the weight of his own body. The thirst is especially increased immediately after the ingestion of food, and also after eating sugar and starchy food. The greater the amount of food and drink ingested the greater the amount of sugar excreted in the urine.

The increased hunger and thirst are not alone the result of increased nutritive changes, but also of disturbances of innervation. In some cases of diabetes, however, hunger and thirst are not increased.

Despite the excessive ingestion of food, the body steadily emaciates the longer the diabetes lasts. The panniculus adiposus disappears, the muscles become flabby and weak. The patients are sometimes confined to the bed for weeks and months on account of exhaustion. They are sometimes reduced almost to skin and bones, while the face not infrequently remains unusually red, almost hectic.

The skin is generally dry and brittle, and often covered with thin, grayish-white scales of epidermis. Local pallor and diminished sensibility (local vascular spasm?) are occasionally noticeable. There is very little tendency to diaphoresis, but hectic sweats may occur if the disease is complicated with advanced pulmonary phthisis. The sweat may or may not contain sugar.

There is often a tendency to obstinate inflammations of the skin

(furunculosis, eczema). Many complain of obstinate pruritus, which interferes with sleep and is sometimes one of the first symptoms of a latent diabetes mellitus. Wounds of the skin are apt to lead to gangrene, and operation wounds heal with difficulty and even become gangrenous. Very marked defluvium capillitii is often observed, and occasionally shedding of the nails. Spontaneous gangrene sometimes sets in in parts of the limbs. All these trophic changes are attributed to overloading of the blood with sugar and other excrementitious products.

Koch mentions, as a constant symptom, enlargement of the peripheral lymphatic glands, as the result of irritation by the sugar-containing lymph. This is not confirmed by my own experience.

It is said that diabetics sometimes pass a larger amount of urine than is accounted for by the fluid ingested, and this has been explained by the aspiration of fluid from the atmosphere through the skin and respiratory mucous membrane. Such aspiration does not take place. It is true that, in a few cases, the amount of urine temporarily exceeds that of the fluid ingested, but this is explained by the withdrawal of water from the tissues.

Pettenkofer and Voit found that diabetics inhaled a diminished amount of oxygen.

The bodily temperature is not infrequently unusually low, and the pulse is generally rapid. Attacks of dyspnœa sometimes occur, either as the result of disease of the heart muscle, or as one of the symptoms of diabetic coma. Œdema may be expected after cachexia has developed.

Psychical changes develop very often. The patients grow fretful, morose, hypochondriacal, and sometimes apathetic. Delirium and maniacal attacks are occasionally observed.

Rheumatoid muscular pains are not infrequent, and neuralgias, particularly of the sciatic, also occur. In rare cases, articular pains with slight swelling are noticed.

Some writers call attention to the frequent occurrence of central facial paralysis. Rosenstein and Maschka noticed frequent absence of the patellar tendon reflex.

Among the organs of special sense, the eye is often affected. The best-known change is the development of cataract which is almost always bilateral, though it may be more marked in one eye. Certain ocular muscles are occasionally paralyzed. There may be diminution in the power of accommodation, and disturbances of refraction from changes in the length of the axis of the globe (often rapidly increasing hypermetropia from shortening of the axis). In three cases, Galezowski observed keratitis attended with severe pain, although the sensibility of the cornea was lost. The vitreous body sometimes presents hemorrhages and opacities. The retina may contain hemorrhages and white patches of degeneration, as in diseases of the heart and kidneys. This may be followed by atrophy of the disk. Among the rarer complications are iritis and mydriasis; amblyopia without ophthalmoscopic changes and hemianopsia are more frequent. The tears sometimes, though not constantly, contain sugar.

The ocular changes, even including cataract, may disappear under proper treatment. The changes in question are attributed to nutritive disturbances

due to the excessive amount of sugar in the blood, and often in the secretions and excretions. This may produce disturbances of function either directly or indirectly, by giving rise to hemorrhages. The cataract has been explained as the result of marasmus and withdrawal of water from the lens by surrounding sugar-containing media, but it also occurs in well-nourished diabetics and usually begins in the central portions which are farthest removed from the surrounding media.

Impairing of hearing, ringing in the ears, diminished power of smell or taste are observed much less frequently. Sugar has been found in the cerumen of the ear.

Pulmonary lesions develop very often in diabetics. Phthisical processes are more frequent, abscess formation is a rarer event. Pulmonary gangrene develops occasionally, but the sputum generally has very little or no gangrenous odor. Many of the patients die of phthisis. The sputum may contain sugar.

The pulmonary changes are probably the result of irritation of the tissues by the blood which is overloaded with sugar and other excrementitious substances. As a rule, the sputum does not differ from that of ordinary phthisis, except that it does not constantly contain tubercle bacilli.

In a case in which the patient suffered from oxaluria, Fuerbringer observed oxaloptysis, and later aspergillus developed in the phthisical foci (pneumonomycosis aspergillus).

The expired air sometimes has a sour, apple-like odor, resulting from the presence of acetone. At the same time the urine generally emits the same odor, and furnishes the ferric chloride reaction.

The circulatory organs are often unaffected for a long time. Leyden has observed cardiac asthma, and Lecorché assumes a diabetic endocarditis. In two of my cases, the blood had an unusually bright red color. The serum may have a cloudy, milky color, from the presence of the finest drops of fat (lipæmia). Its proportion of water may be increased or diminished. Bock and Hoffmann found that it contained 0.3 to 0.35% sugar (normally 0.04 to 0.1%). Petters and Burseri found acetone in the blood.

The majority of patients complain of dryness in the mouth, stickiness of the tongue, and a feeling of constriction in the gullet. The buccal mucous membrane is often dry and sticky to the feel.

The patients also complain frequently of a dry, sour, sometimes sweetish taste. The reaction of the saliva is often acid (lactic acid fermentation of the sugar). This probably explains the rapid caries of the teeth and their loosening. Swelling and bleeding of the gums have been described. The saliva does not always contain sugar. In the later stages of the disease, sprue develops in the mouth, partly on account of increasing marasmus, partly because the sugar in the saliva favors the development of fungi.

The stomach rarely presents functional or other disturbances. According to some writers, the gastric juice contains sugar; others have not confirmed this statement. The liver is sometimes enlarged and tender on pressure.

The bowels are usually constipated, the fæces are hard and dry, and may contain sugar. In one case I found fat in the stools (stearrhœa).

Many patients complain of dysuria, and sometimes of painful sensations in the region of the kidneys. It is also said that a sensation is sometimes experienced as if cold drops were falling into the bladder.

Nocturnal enuresis is frequent in children. Erosions and papillary excrescences are observed in not a few cases near the urethra. Male patients often suffer from inflammatory phimosis. Masses of leptothrix occasionally accumulate in the præputial sac.

Disturbances of the sexual apparatus are very common symptoms. At the outset of the disease, I have repeatedly observed unnatural increase of sexual desire in men. But gradually desire is lost, the testicles become small and flaccid, and complete impotence results.

In Buzzard's patient, the semen contained a few motionless spermatozoa.

Lesions of the external genitalia are frequent in females. In some cases, we find redness and swelling of the labia, with the formation of white plaques which are composed of fungus threads, and are evidently the result of the contact with the saccharine urine. In others, furunculosis of the labia sets in. Phlegmonous processes are especially dangerous on account of their tendency to spread, so that they sometimes extend from the mons veneris to the sacrum and nates. These conditions are the result of nutritive disturbances. Many women suffer from annoying pruritus vaginæ. According to my experience, this is almost always associated with the development of fungi.

Israel observed spontaneous necrosis of the ovary, and, in another case, of the pancreas. Hofmeier states that atrophy of the ovaries is a not infrequent cause of sterility in diabetics. According to Duncan, pregnancy in diabetics is often interrupted by abortion and death of the fœtus.

Emaciation generally occurs; but at the beginning of the disease many patients are well-nourished and even obese.

The average duration of the disease is one to three years. In some cases, death takes place in a few weeks (acute diabetes); in others, the affection has lasted nearly twenty years. As a general thing, it runs a more rapid and dangerous course in children.

Cancer, articular rheumatism, and, according to some, valvular diseases of the heart are said to be rare in diabetics.

Death occurs in many cases from the increasing marasmus, often as a sequel of pulmonary phthisis. The marasmus is accelerated occasionally by cutaneous gangrene following accidental wounds. Death may be the result of cerebral hemorrhage which, according to some writers, is not infrequent in diabetics. In rarer cases, nephritis and anæmic symptoms terminate the scene. This should not be mistaken for diabetic coma, which may assume various forms. In some cases, there is quite sudden unconsciousness, feebleness of the pulse, increasing collapse, and death. In others, there is increasing weakness, followed by headache, restlessness, delirium, maniacal attacks, a feeling of anxiety, difficulty of breathing, increasing cyanosis, feebleness of the pulse, sinking of the bodily temperature, coma, and death. The expired air of such patients often diffuses an intense acetone odor. This condition may last one to five days before death ensues. Finally, cases are observed in which the patients complain of increasing headache, the gait becomes staggering, and then somnolence, increasing coma, and death ensue. In these cases, likewise, there is generally an odor of acetone in the expired air, and the urine gives the ferric chloride reaction. Such conditions may develop in patients whose diabetes had been unrecognized. They have been observed in several cases in which a strict meat diet was suddenly inaugu-

rated. They are susceptible of recovery, although fatal relapses often take place.

Until recently, these conditions were attributed to acetonæmia, but this is rendered very doubtful from the fact that human beings tolerate considerable doses of acetone. Perhaps the symptoms are produced by other unknown excrementitious products in the blood. It has recently been held that diabetic coma is secondary to lipæmia, which results in fatty embolism of the pulmonary and cerebral vessels. According to Frerichs, some cases are the result of weakness of the heart muscle.

III. Anatomical Changes.—The lesions found in autopsies on cases of diabetes are in great part accidental and secondary. If tumors, hemorrhages, softening, or sclerosis are found on the floor of the fourth ventricle, they may be regarded as direct causes of the disease.

The corpse exhibits a tendency to rapid decomposition. Furuncles, ulcers, or gangrenous patches are often seen upon the integument.

The muscles are pale and flaccid, or they have a deep brown color.

Sugar is generally found in any transudations or exudations which may be present in the serous cavities.

Phthisical, purulent, or gangrenous processes may be noticeable in the lungs. Leyden noticed a striking degree of endarteritic changes in those parts which were affected by phthisis.

Frerichs has observed deposits of glycogen in the heart; they are sometimes visible as white patches to the naked eye.

The stomach is not infrequently dilated, its walls thickened, and the mucous membrane congested. Cantani described atrophy of the peptic glands. The gastric contents often emit the odor of acetone. The mesenteric glands may be swollen and congested.

In one case, Buhl noticed gastro-intestinal changes similar to those of Asiatic cholera: distention with fluid, blackish-gray masses, swelling, mucoid degeneration and active desquamation of the epithelium. He is inclined to associate these changes with the symptoms of diabetic coma.

Lesions of the pancreas are not infrequent, and include the following: atrophy, fatty degeneration, necrosis, proliferation of interstitial connective tissue, and formation of calculi, with secondary cystoid degeneration of the excretory ducts.

The spleen is unchanged. The liver often exhibits enlargement, hyperæmia, proliferation of the interstitial tissue, interstitial hemorrhages, more rarely abscesses, calculi, waxy degeneration, atrophy, and portal thrombosis.

The liver cells sometimes contain glycogen many hours after death; this substance is found particularly in the cells at the periphery of the acini. In my own cases, the entire cell presented the glycogen reaction (intense brown color on the addition of iodine and potassium iodide solution). Rindfleisch claims to have seen it confined to the nucleus of the cells. The amount of fat in the liver is sometimes very slight.

The kidneys are often hypertrophic, and may present cysts, waxy degeneration, tubercles, and abscesses. The mucous membrane of the urinary passages may be in a condition of catarrhal inflammation.

Armanni and Ebstein found hyaline degeneration (diabetic swelling of the epithelium) in a large part of Henle's tubes in the cortex (the epithelium con-

verted into transparent vesicles with well stained nuclei); Ebstein also noticed patches of epithelial necrosis in the cortex. Frerichs observed glycogenic degeneration of the renal epithelium, and considers it identical with hyaline degeneration. These lesions are perhaps associated with diabetic coma.

The lesions observed in the central nervous system are meningeal thickenings, adhesions, and hemorrhages. Thickenings of the ependyma of the ventricles are also mentioned. Dilatation of the blood-vessels, atrophy or pigmentation of the ganglion cells, proliferation of the interstitial connective tissue have also been described, but these lesions are accidental, or unimportant.

The sympathetic may be intact or its ganglia, particularly in the solar plexus, may exhibit proliferation of the interstitial connective tissue, pigment degeneration and atrophy of the ganglion cells, and dilatation of the vessels.

In a series of cases, there is no doubt that diabetes mellitus is the result of disturbances of cerebral innervation, the starting-point being found in the floor of the fourth ventricle. It may also be regarded as certain that the liver is concerned in the formation of sugar, and that the sympathetic forms the connecting link between the medulla oblongata and the liver. Hence, diseases of the central nervous system which are associated with vaso-motor disturbances in the hepatic circulation may result in diabetes mellitus. But why the blood becomes overloaded with sugar is not settled with certainty.

The function of the liver cells may be impaired to such an extent that they are unable to convert completely into glycogen the sugar which is conveyed to them from the food through the portal vein; hence the excess of sugar passes into the hepatic vein and thus into the general circulation. Or there may possibly be complete conversion of the sugar derived from the food into glycogen, but the hepatic cells supply the blood with an unusual amount of glycogen and thus of sugar. Or perhaps both processes are combined.

Similar conditions are to be expected if the nervous system is affected, not in the medulla oblongata, but in the sympathetic.

Nor is it impossible that some cases of diabetes mellitus develop in a reflex manner, for example, in peripheral neuralgias.

In addition, there is also a gastro-hepatic form of diabetes mellitus. Clinical experience teaches that the excessive ingestion of sugar and starchy food may give rise to diabetes, either because the liver cannot convert its entire supply of sugar into glycogen, or because the hepatic cells, which are overcharged with glycogen, furnish an excess of sugar to the hepatic veins. The symptoms of diabetes are preceded not infrequently by those of gastro-intestinal catarrh, so that the disease has been attributed to abnormal processes of decomposition and absorption in the gastro-intestinal tract and the portal circulation. Diabetes mellitus sometimes follows diseases of the portal vein or liver itself.

In some cases the source of the sugar has been sought in the muscles. There is no doubt that the muscles form glycogen and sugar, and that in rare cases muscular excretion increases the amount of sugar in the urine in diabetics.

IV. Diagnosis.—A suspicion of diabetes mellitus is often aroused by prominent symptoms. As the sulphate of copper in Trommer's test is also reduced by uric acid, kreatinin, or pyrocatechin, various tests, especially the fermentation test, should be made.

In intermittent diabetes, only certain portions of urine contain sugar, so that, if the symptoms of diabetes are present, but sugar is absent from the urine, it is well to allow the patient to take a hearty meal of sweet farinaceous articles, and to examine the urine two to four hours later.

In mild cases, the sugar disappears on purely animal diet; in severe cases, it merely diminishes.

Abeles and Hoffmann report a case in which an hysterical woman introduced

sugar into the urine and then injected it into the bladder, in order to deceive her physicians.

V. Prognosis.—The prognosis is always grave, and many authors believe that permanent recovery never takes place.

As a general thing, the prognosis is worse in children than in adults, since in the former the disease runs a more rapid and pernicious course.

Advanced emaciation and phthisical changes in the lungs render the prognosis very grave.

Finally, a more serious prognosis attaches to cases in whose etiology heredity plays a part.

VI. Treatment.—Individuals in whose families obesity, gout, or diabetes is hereditary, should avoid sugar and starchy food, and maintain a diet similar to that prescribed in cases of obesity.

After diabetes has developed, strict dietetic rules should be made from the beginning, since they are more important than the administration of drugs. We should endeavor to give the patient as much animal food, especially meat, as possible. A certain amount of variety in the diet is secured by allowing the patient to take all kinds of fats, although this increases, to a very slight extent, the excretion of sugar. Vegetables may also be allowed, but, as a matter of course, only those which contain the smallest amount of sugar and hydrocarbons. As drinks we may recommend carbonated or alkaline waters (Selters, Apollinaris, Vichy, Giesshuebel, etc.), or lemonade made with lactic acid (Aq. destil., ℥ vij.; Acid. lactic., gr. vij.; Natrii bicarbonic., gr. vij., after each meal). Beer, alcohol, sweet wines, and champagne should be avoided; red wines are more serviceable. Ordinary bread should be replaced by gluten bread. Diabetic coma has been known to occur when the patient was suddenly placed on strictly animal diet, so that it is well to begin gradually.

The following is a table of the articles of diet allowed and interdicted:

Articles of food allowed: all kinds of fresh meat, ham, sausage, corned beef, smoked beef, tongue, fowl, and game of all kinds. Fresh and smoked fish, mussels, oysters, lobster, roe, gelatin without sugar, cheese, butter, lard, cod-liver oil, olive oil, cooked green vegetables without sugar (cauliflower, spinach, green tips of asparagus, kohlrabi, beans, white cabbage). Lettuce, endive, horse-radish, water cresses, almonds, nuts, gluten bread, alkaline waters, red wine, white wine, lemonade (with lemons or lactic acid) without sugar, tea, coffee.

Articles of food interdicted: sugar, honey, flour, ordinary bread, farinaceous articles, rice, sago, arrow root, potatoes, maccaroni, oatmeal or barley, milk, whey, chocolate, beer, sweet wines, champagne, alcohol, liquors, sweet or preserved fruits, turnips, onions, radishes, celery, rhubarb, cucumbers, chestnuts.

If the sugar has disappeared for a considerable period, or is present in trifling amounts, one or the other interdicted articles may be allowed for a time. But this may only be done if it does not give rise to an increase in the amount of sugar excreted.

Great importance should be attached to the care of the skin, and the patients should take lukewarm baths several times a day. They should take exercise in the open air, even gymnastics, riding, or mountain tours, if excess is avoided. A trip to the mountains in the summer, and to an uniformly warm climate in the winter is very serviceable, and in some cases is followed by considerable diminution or even disappearance of the sugar in the urine.

The most important medicinal agents are opium, arsenic, salicylic acid, and carbolic acid, but they are useless without anti-diabetic diet.

It is a peculiar fact that diabetics tolerate large doses of opium for a long time without suffering from symptoms of poisoning. Even thirty grains a day have been administered. Morphine has a similar favorable effect, but no certain results are obtained from other narcotics (narceine, narcotine, potassium bromide, strychnine, belladonna, chloral, cannabis indica).

The reports concerning the action of arsenic are less favorable than those concerning that of opium.

Carbolic acid and salicylic acid have been recommended by Ebstein and Mueller, the former drug in solution with peppermint water (gr. xv. [!] : ℥ v.; ʒ i. every two hours), the latter in the form of powder (gr. vij. every hour or two until tinnitus aurium is produced). We must experiment with these remedies, since different patients react differently.

A host of other remedies have been employed, but it will suffice to mention the following: *a.* Alkalies (especially carbonate and bicarbonate of soda) have very little or no effect. *b.* Preparations of ammonia (carbonate, chlorate, acetate). Adamkiewiez recently recommended the acetate very highly. In two of my patients who were treated with carbonate of ammonia, the sugar in the urine disappeared very rapidly, but the pulmonary changes advanced to a speedy fatal termination. *c.* Iodine, iron, quinine. *d.* Creasote, thymol, benzoic acid, iodoform. *e.* Glycerin. *f.* Pilocarpine. *g.* Gall and biliary salts. *h.* Beer yeast, diastase. *i.* Diuretics, drastics, astringents, ergotin. *k.* Galvanization of the cervical sympathetic and medulla oblongata.

A course of treatment at Carlsbad often causes rapid disappearance of the sugar in the urine, but this is probably owing, in great part, to the strict diet. The sugar generally returns after the strictness of the diet is relaxed.

Donkin recommended a strict milk diet, and Dühring claims to have cured (!) a number of cases by the following plan: Three to four meals daily, consisting of ℥ iij. to iv. p. d. of rice, hominy, barley groats, or buckwheat grits, ʒ viij. of smoked or broiled meat, stewed dried apples, plums, or cherries, coffee and milk with wheat bread, and red wine and water after meals.

The various complications of the disease require symptomatic treatment. In diabetic coma, stimulants should be given. Surgical operations must be avoided as much as possible.

Appendix.

Mellituria (glycosuria) is the term applied to the temporary excretion of sugar in the urine. It is merely a symptom of various conditions and rarely requires special treatment. It has been observed under the following circumstances:

a. Excessive ingestion of sugar and farinaceous food.

b. Functional and organic diseases of the nervous system, viz., cerebral hemorrhage, epilepsy, acute delirium, melancholia, general paralysis of the insane, sciatica and other neuralgias, violent mental excitement, cerebral concussion, and meningitis.

c. Disturbances in the pulmonary interchange of gases (?).

d. Cirrhosis of the liver and portal occlusion, especially after the ingestion of sugar and farinaceous foods, probably because the sugar which has been absorbed passes directly into the general circulation without being converted into glycogen.

e. Convalescence from infectious diseases (cholera, variola, pneumonia, malaria, erysipelas, phlegmonous inflammations, etc.).

f. After poisoning with carbonic oxide and illuminating gas, glycosuria is

sometimes observed for several hours. Whether the reducing substance found in the urine after opium and chloral poisoning is really sugar requires further investigation. The reducing substance found in the urine after nitrobenzol poisoning is not sugar. Arsenic poisoning and prussic-acid poisoning produce true glycosuria with increased thirst and increased amount of urine.

g. Sugar is found in the urine of puerperal women, especially if they do not nurse, and in that of infants. Hoffmeister and Kaltenbach showed that milk sugar is found in such cases (lactosuria).

h. According to Liveing, glycosuria is not very rare in chronic eczema.

4. *Diabetes Insipidus.*

I. Etiology.—Diabetes insipidus is an independent disease whose chief symptoms are increased excretion of urine (polyuria) and increased thirst (polydipsia), the latter being secondary to the former.

The disease is rarer than diabetes mellitus, and affects men two or three times as often as women.

Middle life (fifteen to forty-five years) is attacked most frequently. Kuelz recently collected thirty-five cases in children; two of these cases began in the first year of life.

The disease is sometimes hereditary, being observed in several children of one family, or in several generations. Diabetes mellitus and diabetes insipidus may also occur in different members of the same family. The disease appears occasionally in families which are also affected by insanity and nervous diseases.

It is produced not infrequently by diseases of the nervous system, such as concussion of the nervous system, penetrating wounds of the skull, inflammations of the meninges, hemorrhage, inflammation, softening, and tumors of the central nervous system, hydrocephalus, chronic diseases of the spinal cord, and neuroses (hysteria, Basedow's disease, chorea, epilepsy). Diabetes insipidus may be looked for with so much more certainty the more the floor of the fourth ventricle has been affected.

Bernard showed that injury to the floor of the fourth ventricle, above the site whose irritation causes diabetes, will give rise to increased excretion of urine. According to later experimenters, irritation of adjacent parts produces the same effect. Polyuria is produced in rabbits by irritation of the vermis of the cerebellum, in dogs by section of the splanchnic nerves; also after section of the spinal cord below the twelfth vertebra.

Diabetes insipidus is sometimes attributed to mental and bodily strain, fright, insolation, cold, exposure to wet, and taking cold drinks when the body is heated, but such statements are not always well founded.

It sometimes follows infectious diseases (intermittent fever, diphtheria, scarlatina). When observed in syphilis, it is generally the result of softening or gumma of the central nervous system.

II. Symptoms.—The symptoms sometimes develop within a few hours after the action of the etiological factor. In other cases, they develop so gradually that it is difficult to determine the onset of the disease.

The most constant symptom is the increased excretion of urine, and two to five times the normal amount may be passed. Trousseau reports a case in which forty-three litres were passed daily. The patients are compelled to micturate frequently at night. The smaller the capacity of the bladder the more frequently is the urine voided, and the smaller the quantity discharged at one time. The urine is light-yellow, sometimes

almost as clear as water, has an acid reaction, but readily becomes neutral or alkaline after standing, and has a low specific gravity (1005–1008, occasionally even 1001 or 1000.5).

As a rule, the solid constituents in the entire daily amount of urine are increased.

The amount of urea is generally increased: Senator has found as much as two and one-half ounces per diem. In a doubtful case, reported by Hoffmann, the uric acid was replaced by hippuric acid. According to Senator, the excretion of kreatinin is unchanged. The phosphates, chlorides, and sulphates are generally increased. These changes are explained, in great measure, by the copious supply of water to the tissues, as the result of the increased thirst. To this is probably owing the fact that inosit has been found in the urine in a number of cases. Strauss showed that this also appeared in the urine of healthy individuals after copious draughts of water. Temporary glycosuria may occur; albuminuria is rare.

An increased feeling of thirst is a constant symptom. The greater the thirst the greater the amount of urine excreted, though these two factors do not correspond entirely at all times. For a short time, the amount of urine may exceed the amount of fluid ingested, as the result of withdrawal of water from the tissues. If healthy and diabetic individuals drink equal amounts of fluid, the amount of urine is increased more rapidly in the former, but persists in the latter for a longer time, inasmuch as the excretion of urine occurs more uniformly.

The increased feeling of thirst produces a sensation of dryness and stickiness in the mouth and throat, and occasionally a feeling of constriction in the pharynx. Children sometimes drink their own urine or any other fluid which falls into their hands.

That polydipsia is the result of polyuria is evident from the fact that the latter continues after the patients are deprived of fluids. In very rare cases, however, there appears to develop a primary polydipsia with secondary diabetes. Nothnagel recently reported the following case: A mason, æt. 35 years, fell on the back of the head; unconsciousness; half-hour later, intolerable thirst; two and a half to three hours later, permanently increased excretion of urine. In primary polydipsia, the amount of urine must diminish very rapidly and become subnormal if the patients are deprived of fluids.

The insensible perspiration is usually diminished, but occasionally unchanged. The skin is very dry and brittle. Some patients complain of paræsthesiæ and pruritus; furunculosis is rare. Hyperhidrosis and salivation have been observed in exceptional cases.

The bodily temperature may be abnormally low, and the patients grow cold very quickly, owing to the fact that a large amount of heat is given off in order to warm the large amounts of fluid ingested.

If the disease develops in early childhood, the bodily development is often very much retarded. In other cases, general nutrition is very little impaired. The appetite is more frequently diminished than increased. There is sometimes a desire for peculiar articles of diet. Gastric pressure, flatulence, eructations, and intestinal disturbances are not uncommon.

Nervous symptoms (pressure in the head, vertigo, depression, etc.) occasionally develop. Paralysis of cerebral nerves sometimes occurs, with relative frequency in the abducens.

The following ocular changes have been observed: retinal hemor-

rhages, neuroretinitis with fatty degeneration as in Bright's disease, atrophy of the optic nerve, hemianopsia, and amblyopia; cataract does not develop.

The disease may last many years (fifty years in Willis' case). Remissions and exacerbations are not infrequent; the latter occur particularly after emotional excitement. The symptoms sometimes disappear during intercurrent diseases, but return at a later period. Diabetes insipidus occasionally appears at the beginning or end of diabetes mellitus. Death is the result of intercurrent diseases, increasing marasmus, or the advance of the primary disease.

III. ANATOMICAL CHANGES.—There are no specific anatomical changes in this disease. Connective-tissue proliferation and degeneration of the solar plexus have been found. The kidneys are sometimes enlarged and contain an unusual amount of blood; Neuffer noticed dilatation of the urinary tubules and fatty degeneration of the epithelium.

The nature of the disease is unknown, but the symptoms are probably the result of vaso-motor disturbances in the renal circulation, in the production of which the sympathetic renal fibres play the chief part.

IV. DIAGNOSIS.—The diagnosis is made from the increased excretion of urine of low specific gravity, and the increased feeling of thirst. The following conditions must be taken into consideration in differential diagnosis :

a. In diabetes mellitus, the specific gravity of the urine is increased, and sugar is present.

b. Cirrhosis of the kidneys causes increased thirst and increase in the amount of urine with diminished specific gravity, but albuminuria and left ventricular hypertrophy are also present.

c. Primary polydipsia with secondary polyuria is distinguished by the effect of the withdrawal of fluids upon the amount of urine.

d. Transitory and symptomatic polyuria is only of short duration.

Transitory polyuria is observed under the following conditions : after the onset of cerebral apoplexy, after mental strain in hysteria, and during convalescence from severe infectious diseases, especially typhoid fever. I observed it twice after the administration of digitalis, once after salicylic acid. In some individuals (especially females) it occurs after coitus. It is also observed frequently in diseases of the urinary passages.

V. PROGNOSIS.—Permanent recovery from diabetes insipidus occurs only in exceptional cases, but life is often maintained for a long time. The prognosis is so much more serious the more rapid the emaciation and the more grave the primary disease.

VI. TREATMENT.—Causal indications must first be met. In a patient æt. 6 years, in whom the disease was associated with syphilis, Demme obtained successful results by means of the inunction treatment. If there is marked anæmia, I have obtained great benefit from iron preparations, notably tinct. ferri acetic. and tinct. ferri chlorat. æther. (3 i. three or four times a day).

If there are no causal indications, we would recommend the administration of opium and lead in combination (Plumb. acet., gr. $\frac{3}{4}$; Opii. puri, gr. ss. ; Sacch. alb., gr. vij., one powder every three hours). As in diabetes mellitus, very large doses of opium often are tolerated. The thirst may be quenched with acid drinks ; the patients should wear thin flannel to avoid catching cold.

The other remedies employed include the following: *a.* Valerian, potassium bromide, ergotin, arsenic, belladonna, digitalis, castoreum, asafœtida. *b.* Jaborandi and pilocarpin. *c.* Creasote, carbolic acid, sodium salicylate. *d.* Tannin, turpentine, copaiba, potassium iodide, calomel, nitric acid. *e.* Constant galvanic current to the spinal cord and renal region, also to the medulla oblongata, cervical cord, pneumogastric, and sympathetic.

5. *Rickets. Rachitis.*

I. Etiology.—The nutritive changes which lie at the bottom of rachitis are manifested chiefly by abnormalities in the growth of the bones. Proliferation of the cartilaginous and periosteal portions, with imperfect and irregular calcification, produces deformities of the bones which are such a prominent part of the clinical history that we might almost be tempted to consider the disease a purely local affection of the bones.

Rickets occurs most frequently between the ages of seven to thirty months, is rare beyond the third year, and hardly ever occurs beyond the age of five years.

In a number of cases, the bone changes develop in utero (fœtal rachitis). But it is not certain that these changes are histologically identical with the rachitic process.

The term congenital rachitis is applied to those cases in which the disease appears very soon after birth.

Some authors assume the existence of rachitis tarda, in which the disease develops during the period of puberty, but this assumption is still unproven.

The disease is more common among the children of the poorer classes.

It is an extremely frequent disease of childhood. According to Ritter v. Rittershain, about thirty per cent of the children brought to the Prague Policlinic presented evidences of rachitis.

It is generally the result of the combination of a number of causes. Hereditary agencies are often at fault. Rachitic children are often born of parents who suffer from phthisis or the late stages of syphilis or exhausting diseases in general. Children born when the parents have attained an advanced age are attacked not infrequently with rickets. Rapid succession of children, or nursing by a pregnant mother, or excessive lactation also favors its outbreak. Whether rachitis as such is hereditary has not been proven with certainty. It has also been asserted that anæmia of the mother favors the development of the disease.

In many cases, the disposition to rachitis seems to be acquired, for example, as the result of improper nourishment. The disease very often attacks children who are fed with cow's or goat's milk, condensed milk, infant foods, or a diet which is rich in vegetables. It is more apt to develop if the patients, in addition, live in overcrowded, dark, damp rooms. The exciting cause is sometimes furnished by intercurrent diseases.

The disease is extremely frequent in England, Holland, France, and Germany, while it is extremely rare in the tropics. It is said not to develop beyond the height of 3,000 feet above the sea level.

II. Symptoms.—In some cases, rachitis begins without special pro-

dromata. Children who had learned to walk tire very easily, then cease to walk, and present deformities of the limbs. Or disturbances are observed in the outbreak of the teeth. The children attain the age of two years before a tooth appears, or the eruption of the teeth is irregular, or dentition ceases. Finally, deformities of the skull or spinal curvatures may be noticed. In other cases, the disease is preceded by prodromata, consisting of obstinate disturbances of gastro-intestinal digestion. The appetite is disturbed (generally anorexia, more rarely boulimia), the tongue is almost always coated, there is a sour smell from the mouth, eructations and vomiting are frequent, the abdomen is tympanitic, and there is obstinate and generally foul-smelling diarrhœa. General nutrition is also impaired. The face grows pale, the muscles flabby, the panniculus adiposus emaciates, and sinuous veins appear beneath the skin. After these symptoms have lasted for a longer or shorter time, the characteristic changes in the bones make their appearance.

The entire skeleton is generally affected, the changes beginning often in the skull, and then extending to the trunk and limbs. More rarely the process begins in the lower limbs and extends upwards, and the skull is then relatively unaffected. This is most apt to happen if the disease begins after the fifteenth to eighteenth month. The rachitic changes are often developed with striking similarity in corresponding parts of the body.

Rachitis of the skull is characterized by the peculiar shape of the head (vide Fig. 12). While the skull appears to have increased considerably in size, the face appears very much smaller. The long diameter of the head is often increased (dolichocephaly). The occipital bone is flattened, the frontal and parietal eminences are unusually prominent as the result of subperiosteal proliferations.

The frontal bone passes straight upwards, the parietal bones project outwards. On transverse section the skull is almost quadrilateral. But the increase in the size of the skull is only apparent, and in reality the skull is smaller than normal if compared with that of healthy children of the same age.

Another important symptom is the patency of the fontanelles and often of the sutures, and elevations along the edges of the bones. The large fontanelle, which should be ossified by the middle of the second year, remains soft and compressible. It is sometimes increased in size, extending anteriorly to the middle of the frontal bone, posteriorly to that of the parietal bones, and laterally to the frontal eminences. Its four sides are bounded by convex, prominent edges of bone. From this fontanelle we may occasionally trace all the sutures, which also appear broadened; the adjacent edges of bones are elevated like a wall.

In severe cases, craniotabes is usually noticeable. The occiput, particularly near the lambdoidal suture, is as thin as parchment and crackles, or such places are situated only here and there, or in places the bony substance has disappeared entirely and the dura mater and pericranium are in close contact with one another. As many as thirty openings of this kind have been found in the occiput. Their development is probably the result of the combination of various causes: pressure of the brain on the occiput with counterpressure of the pillow; constant lying in bed, and irregular absorption processes of the osseous substance with defective new-formation of bone. Excessive pressure upon such places may give rise to unconsciousness, general convulsions, or spasm of the glottis. Similar softened and atrophied portions of bone are found occasionally

in the parietal, sphenoid, and even the frontal bone. Slight grades of this condition are said to be observed at times in non-rachitic children.

The hair of the scalp, especially over the occiput, is apt to fall out, on account of the profuse sweating and the constant recumbent position. The hairs become brittle and break, finally they fall out, and the occiput then appears more or less bald.

Fig. 12.

Rachitic child, æt. 2 years, with rachitic skull and thickenings of the costal cartilages and epiphyses.

The systolic cerebral murmur is a blowing, vascular sound, coincident with cardiac systole, and which is generally heard with greatest distinctness over the large fontanelle, but occasionally over the parietal bone and lesser fontanelle. This was erroneously held to be characteristic of the rachitic skull. Juracz has shown that it is observed in children from the age of three months to the end of the sixth year. It occurs only when a similar sound is heard in the internal carotid, whence it is conveyed to the surface of the brain. According to Juracz,

the murmur is the result of stenosis of the carotid, owing to the temporary diminished growth of the carotid canal.

The symptoms of rickets of the jaw assume a prominent part in the clinical history of rickets of the skull.

The lower jaw approaches the shape of a hexagon, inasmuch as the anterior portion corresponding to the incisors becomes flattened, while the lateral portions behind the canines are bent at an angle posteriorly. At the same time the alveolar process of the jaw turns, its upper surface being directed more posteriorly and internally, its base more externally. As a matter of course, this induces a defective position of the teeth. Fleischmann attributes these changes to the traction of the muscles inserted into the lower jaw, the bone itself being abnormally flexible. He also attributes to similar conditions the changes in the shape of the superior maxilla. This bone is narrowed laterally in the region of the malar process, so that its long axis increases in size, and it becomes beak-shaped.

If rachitis develops before the seventh month, dentition often remains absent, so that the children may attain the age of three years with toothless jaws. In other cases, the eruption of the teeth is delayed and irregular, or they may be situated in abnormal situations, for example, they perforate the anterior alveolar wall. They often grow prematurely carious and loose. Nicati calls attention to the fact that terraces which are in places deprived of enamel, form upon the persisting incisor teeth.

In normal dentition, the first teeth generally appear in the seventh month. Twenty teeth appear by the end of the second year, four incisors, two canines, and four molars in each jaw.

At the end of the fourth year two permanent molars appear, so that the jaw contains twenty-four teeth. At the end of the seventh year, two new molars in the upper and lower jaws, making twenty-eight in all. Between the eighteenth and thirtieth years, the four wisdom teeth make their appearance.

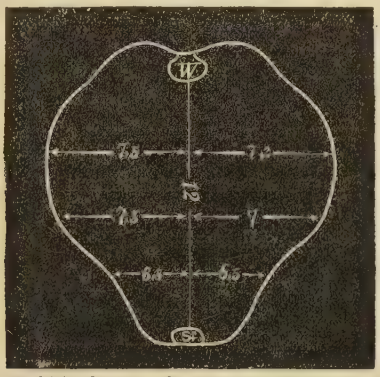
During the seventh year and later, the milk teeth are replaced by the permanent teeth. The former fall out in about the same order in which they appeared.

Among the symptoms of rickets of the thorax, the chief attention is attracted by the button-shaped enlargements at the boundary between the ribs and costal cartilages. In lean individuals, they are distinctly visible as prominences; in others, they are readily felt with the fingers. They form a curve running from above internally, below and externally (rachitic rosary). They are the result of proliferation of the cartilage cells, and are co-ordinate with the epiphyseal enlargements at the ends of the bones of the extremities. On account of the yielding character of the ribs, the entire thorax is deformed. At first it undergoes flattening, and finally is depressed in the lateral regions. This depression begins between the fifth and seventh ribs, and then extends upwards and downwards. The transition from the posterior to the anterior portions of the ribs is sudden and angular. The lower edge of the ribs projects outwardly, and is, to a certain extent, pushed upwards, so that the long diameter of the thorax is shortened. Retractions are noticeable with each inspiration. The sternum often projects at an acute angle (chicken breast). On transverse section, the shape of the thorax resembles that of a pear (vide Fig. 13).

The shape of the rachitic thorax is the result of various factors. In the first

place, the soft ribs yield to the inspiratory traction of the lungs inwards, especially since the disease is often complicated by obstinate and extensive bronchitis. In addition, the ends of the ribs, on account of their changed growth, grow anteriorly past the enlarged costal cartilages and thus favor a bending inwards (Hueter). Nor should we underestimate the effect of lifting the children up by means of the hands pressed beneath the lateral surfaces of the thorax.

Fig. 13.



Transverse section of the rachitic thorax. *St*, sternum; *W*, vertebral column. The numbers indicate centimetres. One-fourth natural size.

The ribs sometimes present fractures or subperiosteal enlargements. Finally, a prominence, possessing a very acute angle, may develop between the manubrium and the body of the sternum, and the latter may form a more or less deep furrow.

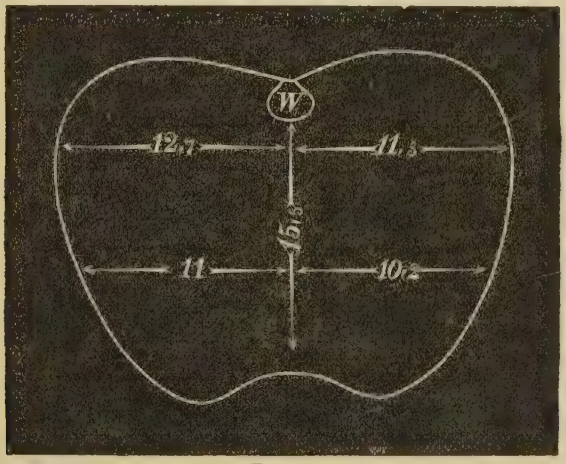


Fig. 14.

Transverse section of a rachitic thorax, with a furrow-shaped depression of the sternum. *W*, spine; *St*, sternum. At the level of the fifth costal cartilage. The numbers indicate centimetres. One-fourth natural size.

The clavicles often present rachitic changes. Both epiphyses are enlarged into shapeless masses, the gentle curves are replaced by angular flexions, and partial, even complete fractures may ensue, generally as the result of vigorous pressure with the arms. The scapulæ may also present thickening of the free border, sometimes partial fracture of the lower half.

Thoracic deformities are greatly increased after rachitic spinal curvatures supervene. Kyphosis is most frequent, lordosis or scoliosis is rarer. The curvature is generally most marked at the level of the first lumbar vertebra, but often involves the adjacent dorsal and lumbar vertebræ. Kyphosis, lordosis, and scoliosis are often combined.

The pelvis not infrequently presents the characteristics of the flat, rachitic pelvis, the sacrum being pushed into the pelvic cavity, as it were, by the weight of the body. The conjugate axis is very small, and in female patients this may become a source of difficult labor. In addition, the acetabular region is occasionally pushed inwards, so that the transverse section of the pelvis becomes heart-shaped. These deformities are produced not alone by the weight of the body, but also by the traction of the muscles inserted into the pelvis.

The most noticeable change in the bones of the limbs is the enlargement of the epiphyses, particularly at the lower end of the ulna and radius, tibia and fibula (vide Fig. 12). A deep groove often forms beneath them, and separates the epiphyseal enlargements from the wrist and ankle joints. In addition, there are curvatures of the bones which are generally exaggerations of the normal curves. In the legs, as a rule, the curvature is convex to the outside, more rarely towards the front, rear, or inside. The forearm is generally curved convex towards the extensor side. The humerus and femur are often curved, but to a less degree. The epiphyses are often pushed strongly to one side of t e shaft of the bone. These deformities are the result of the action of the weight of the body, together with muscular traction. Partial fractures sometimes occur upon the convex side of the curvatures; complete fractures occur more rarely. The former affect mainly the bones of the forearm and leg, the latter the humerus and femur. The patient's gait becomes awkward and waddling.

Many of the children manifest precocious mental development, probably owing to the fact that, on account of the restriction of bodily movements, they are more confined to mental exercise. They sometimes complain of pain in the limbs, and this appears either spontaneously or as the result of pressure on the diseased bones. There is often obstinate bronchitis which does not yield until the rachitis is cured, and often terminates in broncho-pneumonia. The heart may be displaced on account of the deformity of the thorax and spine. Leucocytosis, nucleated or red globules, and diminution of the number of red blood-globules have been described. Enlargement of the spleen is not uncommon. In a number of my cases the spleen projected more than six centimetres beyond the left ribs. Some physicians claim to have found enlargement of the liver. The appetite is generally poor, but when symptoms of tabes mesenterica appear, there is usually insatiable boulimia. Disturbances of digestion are almost constant. Chemical examination of the fæces has shown an increased amount of lime, but not of phosphoric acid. There are no characteristic urinary changes; the amount and specific gravity of the urine present notable variations.

The results of chemical examination of the urine are in part contradictory. According to recent investigations, the amount of lime in the urine is unchanged or diminished.

The general development of the body is retarded in this disease. The children often sweat profusely upon the head and neck, and experience

a sensation of increased heat under the bed-clothes, so that they are apt to lie uncovered at night.

The disease generally lasts several months. Acute rachitis (sudden onset and rapid course of a few weeks) has been described, but the symptomatology is so different from the typical clinical history of rickets that great care must be exercised in the interpretation of such cases.

Complications are frequent. The disease is very often associated with scrofula, but we do not believe that rickets, per se, gives rise to enlargement of the peripheral lymphatic glands. The scrofula may be followed by phthisical changes in the lungs and miliary tuberculosis. Spasm of the glottis often occurs in rachitic children; chronic hydrocephalus and eclamptic attacks are also observed. Waxy degeneration sometimes develops. According to Rehn, rickets may be associated with osteomalacia. Cortical cataract has also been observed in rachitic children.

A large proportion of the cases are cured by proper treatment. Even considerable deformity of the limbs may disappear after a certain period, but the patients sometimes remain dwarfish. Supernumerary bones develop not infrequently in the cranial sutures, and the fontanelles and sutures appear very deep. The bones are sometimes very much condensed (rachitic sclerosis or eburnation), in later years they may become extremely brittle. The patients are placed in special danger by spasm of the glottis, bronchitis, broncho-pneumonia, exhaustion from obstinate diarrhœa, or tubercular processes.

III. Anatomical Changes.—The bones are affected almost exclusively, the internal organs remaining free from specific lesions. The following unimportant changes have been found: milk patches beneath the anterior epicardium (probably from friction against the enlarged costal cartilages), enlargement of the spleen (dependent mainly on hyperplasia of the cellular elements), accumulation of fat in the liver.

The rachitic bones present the following lesions: enlargement of the epiphyses, thickening and hyperplasia of the subperiosteal layers, unusual congestion of these parts, flexibility and softness of the bones. The bones, especially those of the skull, may not infrequently be cut with a knife. If a longitudinal section is examined, it is seen that the epiphyseal changes start from the cartilage situated between the epiphysis and diaphysis. This part, as is well known, provides for the longitudinal growth of the bone by the constant production of cartilage cells, which are added to the diaphysis and are converted into osseous tissue.

The healthy epiphyseal cartilage is divided into the epiphyseal and diaphyseal zones. The former has a bluish-white color, and is one to two millimetres in height. It is subdivided into two parts. In the younger one, adjacent to the epiphysis, the process consists chiefly of proliferation of cartilage cells and gradual arrangement in longitudinal rows (hyperplastic portion), while in the older one, nearest to the diaphysis, there is chiefly an increase in the size of the cartilage cells (hypertrophic portion). The diaphyseal portion of the epiphyseal cartilage is about 0.5 mm. in height, and has a yellowish color. It is also known as the zone of temporary calcareous infiltration, because the cartilage in it is gradually converted into osseous tissue. Both zones are sharply separated from one another by a straight line.

In rickets, both portions of the epiphyseal cartilage are greatly increased in height. The proliferation zone may be several centimetres in

thickness, and project laterally from the side of the bone. The sharp boundary between the two layers of the epiphyseal cartilage is lost, and both are abnormally vascular. In the zone of temporary calcareous infiltration, the vessels are increased in size and number, and extend into the proliferation layer of the epiphyseal cartilage, which under normal conditions contains no vascular spaces. In the healthy cartilage, the calcification of the diaphyseal portion occurs in an uniform manner; in rickets, we find irregular patches infiltrated with lime which, in places, extends into the proliferation layer. In this manner the diaphyseal portion assumes a porous, spongy character (spongoid tissue).

The growth of bone in thickness depends upon the periosteum. On the surface of the periosteum adjacent to the bone proliferations form, and are gradually converted into osseous tissue. At the same time an absorption of osseous tissue occurs on the side of the medullary cavity. In rickets, periosteal growth undergoes changes similar to the epiphyseal growth. There is considerable increase of the proliferation layer, which may attain a thickness of several millimetres. This layer is very rich in vessels. Calcification occurs irregularly in islets, so that a spongoid tissue is formed. Osteoid tissue often adheres to the inner surface of the periosteum, when the latter is stripped from the bone. At the same time the medullary cavity continues the process of absorption, even to an excessive degree, so that the bone becomes unusually flexible. The medulla is generally very red, and occasionally has a lymphoid appearance.

The microscopical changes are still the subject of dispute. The proliferation layer presents excessive proliferation of the cartilage cells. The cell groups contain an unusually large number of closely aggregated cells, between which the basement substance has disappeared in great part. The latter loses its homogeneous character and assumes a more fibrillated structure.

In the zone of temporary calcareous infiltration, the development of medullary spaces, and of vascular spaces within them, is unusually extensive; contrary to the rule, this often extends into the proliferation zone. Calcification and ossification occur irregularly, and also extend into the proliferation zone. Some of the cartilage cells are converted directly into bone corpuscles. Klebs found that others were converted into medulla cells, and then into connective-tissue corpuscles, so that the vascular spaces in the medullary spaces were surrounded in many places by dense masses of connective tissue. Similar processes are observed in the periosteum.

The specific gravity of the bones is diminished. In a child æt. 8 years, Trousseau found that the entire skeleton weighed one kilogram (normal weight, seven to eight kilograms).

Friedleben found an increased amount of water, fat, and carbonic acid in the bones, diminution of lime salts; in general, increase of organic, diminution of inorganic constituents.

Concerning rachitic changes in bones, two factors must be distinguished, viz.: the proliferative processes and excessive vascularization, and the irregular and imperfect calcification.

It is evident that a deficiency of lime salts in the bones may arise when the food is poor in these salts, or their absorption from the intestines is interfered with, or if there are conditions of the blood or bones which prevent the precipitation of lime salts from the blood.

A deficiency of lime salts in the food does not obtain in the majority of cases, because milk contains sufficient amounts for the development of bone. It has been claimed that rachitic changes may be produced experimentally in animals by depriving them of lime salts, but this has been denied.

From the fact that obstinate diarrhœa is a prodrome of rachitis in many cases, it was assumed that lactic acid was formed by the fermentation of ingested milk, that this, being absorbed by the blood and tissue juices, dissolved the lime salts and prevented their precipitation in the bones. Great importance was attached

to the fact that lactic acid was said to be demonstrable in the urine. But this has recently been sought for in vain in the urine, and it has never been found in the bones. Nor does the urine contain an unusually large amount of lime salts. Senator has recently called attention to the view that the carbonic acid in the tissues perhaps acts as a solvent of the lime salts. Seemann believes that the absorption of lime salts from the intestines is diminished, and this is rendered plausible by the abundance of lime in the fæces. This writer calls attention to the large amount of the potassium combinations in milk (particularly of animals), and to a still greater degree in vegetable food. When the potassium salts are absorbed they combine with all the chlorine at their disposal, so that very small amounts of chlorine remain to aid the absorption of lime salts. Hence, the latter pass in the fæces, in part unchanged.

But this factor explains only the poverty of the bones in lime, not the inflammatory character of the proliferatian of cartilage cells and vessels. In our opinion, rachitis may be explained as follows: disturbances of nutrition in consequence of perverse nutritive processes or other general causes; a predominantly local inflammatory affection of the epiphyseal cartilage; imperfect and irregular calcification, on account of deficiency of lime in the tissues and the existing inflammatory conditions in the cartilage. This theory tallies with the experiments of Wegner, who produced inflammations of the epiphyseal cartilages in animals by administration of phosphorus, and at the same time gave to them food deficient in lime salts. This experiment was followed by rachitic changes in the bones.

IV. Diagnosis.—The diagnosis is easy. Delayed and irregular dentition and profuse sweating of the head and obstinate diarrhœa point towards latent rickets.

Osteomalacia occurs almost exclusively in adults. In cases of congenital syphilis, the epiphyseal cartilages may become separated, but the patients are only a few weeks old, and there are other evidences of syphilis on the skin and mucous membranes. The rachitic skull should not be mistaken for chronic hydrocephalus. In the latter, spasms often occur, and mental development is imperfect.

V. Prognosis.—The prognosis is not unfavorable as regards life, if the disease is not too far advanced, or associated with scrofula, tabes mesenterica, or other serious complications. Deformities of the bones may subside spontaneously. Persistent deformities of the thorax and spine may give rise to shortness of breath and a tendency to inflammations of the air passages.

VI. Treatment.—Proper nourishment and treatment of diarrhœa are extremely important as prophylactic measures. In our own experience, the following plan of treatment has been attended by the most rapid and successful results in this disease:

The diet should be regulated; infants should be nursed at the breast, if possible, or should receive cow's milk with the addition of lime-water; older children should receive less vegetable, more animal food.

The children should be kept a good deal in the open air, and should sleep upon a hard mattress. They should not be urged to walk, and in carrying them care should be taken not to produce artificial deformities.

A teaspoonful of cod-liver oil is given morning and evening. For four weeks, a sodium-chloride bath should be given every morning (28° R.; sodium chloride, ℥ xxx.–l.; twenty minutes' duration; then one-half to one hour's rest in bed).

The following prescription may be ordered:

℞ Ferri lactic.,
Calcar. phosphoric..... āā 3 iij.
Magnes. carbon.,
Natr. chlorat.,
Sacch. alb..... āā 3 iss.
M. D. S. The point of a knife full t. i. d. after meals.

Diarrhœa offers no contra-indication to this plan of treatment.

Preparations of iron and lime, bitters, tonics, phosphorus, and arsenic have also been recommended in rickets.

6. *Softening of the Bones. Osteomalacia.*

I. Etiology.—Osteomalacia is a rare disease, hardly more than one hundred and seventy cases having been reported hitherto. It is most frequent between the ages of 20–50 years, but is not as rare in childhood as has been believed. Among one hundred and thirty-one cases collected by Litzmann, men were attacked eleven times, women one hundred and twenty times. An extremely large number of the cases are associated with pregnancy and parturition. It occurs particularly in certain localities, such as the Rhine districts, East Flanders, and in the vicinity of Milan. Casati observed it, in Milan, in 0.8 per cent of all puerperal women. The majority of the patients came from the Olona valley, in which typhus fever and pellagra are prevalent.

The non-puerperal form is sometimes attributed to colds, wetting, insufficient food, and dark, damp apartments. Rehn described a combination of osteomalacia and rickets in children, but states that the former is not connected with congenital syphilis.

II. Symptoms.—The first symptoms consist of rheumatoid pains in those parts of the skeleton which are first affected—in the puerperal form, generally in the pelvis; in the non-puerperal form, in the spine. The pains are sometimes worse at night and cease after profuse sweating, or they are increased after prolonged sitting, on motion or pressure. Febrile movement is not infrequently present. Curvature of the bones soon occurs. The promontory of the sacrum passes deeply into the pelvic cavity; the acetabula are bent inwards. The rami of the pubis project like a beak, and the transverse section of the pelvis assumes a cordate shape. The narrowing of the pelvis may not alone interfere with labor, but also with the evacuation of the bladder and rectum. Individuals suffering from softening of the pelvis complain very quickly, while sitting, of pain in the tubera ischii. The normal curves of the spine are morbidly increased. The cervical curve is sometimes so great that the chin and sternum come in contact. Considerable deformity appears in the lumbar spine. The spine is shortened, and the patient sometimes shrinks to dwarfish dimensions. The ribs and sternum often present serious deformities, and these may be associated with partial or complete fractures. Hence compression and displacement of the lungs and heart, palpitation, dyspnœa, and asthmatic attacks. The limbs may also be subject to curvature and fracture, and the gait becomes waddling and finally impossible. Multiple fractures after slight injury are sometimes the first evidence of osteomalacia. In fractures, the formation of callus may be absent or incomplete, or it may undergo absorption at a later period. Softening rarely occurs in the bones of the skull. The teeth are not attacked, although they may become carious and drop out.

The muscles are soft and flabby. Fibrillary twitchings, spasms, and painful contractures have been described. They occur spontaneously or after slight cutaneous irritation.

The sweat, saliva, and milk are said to be overloaded with lime salts, and the latter are also said to be excreted through the bronchial and gastro-intestinal mucous membrane (?).

No specific changes are noticed in the urine. There is sometimes temporary increase of uric acid. The specific gravity is generally diminished. As a rule, the excretion of urea and phosphoric acid is diminished. There is no constant increase in the excretion of lime salts. Lactic acid has been found repeatedly in the urine (also in normal urine). Albumin and hemialbumose (vide Vol. II., page 245) are found occasionally.

The urine often contains a sediment of carbonate, phosphate, and oxalate of lime, and the kidneys may contain concretions of a similar character.

Leube found 0.345 gm. lime in the fæces in two days.

The disease generally lasts many years. The most acute case lasted nine months; the most protracted, thirteen years. Remissions and exacerbations are frequent, the latter generally during pregnancy. Death occurs from increasing marasmus, from respiratory and circulatory disturbances caused by the deformity of the thorax. Recovery is rare.

III. Anatomical Changes. — The principal changes affect the bones. They are sometimes as flexible as if the lime salts had been removed with acids, or they form membranous structures like the intestines, and are readily cut with a knife.

The medullary cavity is increased in size. The osseous parts of the spongy substance may have disappeared, so that it forms a continuous mass of medulla. If the absorption of the bony framework has occurred only in places, cystic medullary spaces are produced. The medulla itself is at first congested and here and there contains extravasations of blood. In later stages it contains a large amount of fat, and becomes yellow, finally atrophic and gray.

The Haversian canals are increased markedly in size and filled with reddish succulent tissue. The bone thus appears porous and unusually juicy. While the medullary cavity and Haversian canals increase in size, the surrounding osseous substance atrophies, the latter process occurring from the medullary cavity towards the periosteum. Finally, only a thin layer is left beneath the periosteum, and even this may disappear almost entirely. The periosteum is thickened, its proliferation layer congested and contains extravasations. According as the remaining masses of bone are flexible or still contain firm portions, the diseased bone presents a varying tendency to fracture.

The microscope shows great changes in the osseous tissue in the immediate vicinity of the medullary cavity and Haversian canals. This part stains readily with carmine, has lost the lime salts and assumed a fibrillated structure, and contains a few spindle-shaped, unbranched remains of bone-corpuscles. At a later period gradual mucoid liquefaction and absorption seem to take place. At the boundary between the healthy and diseased tissues are found the so-called Howship's lacunæ, in which myeloplaques have been found.

The medulla of the bones and the contents of the Haversian canals present congestion of the vessels (passive hyperæmia, according to Rindfleisch) and numerous extravasations. In the red marrow we find only the remains of fat cells, in great part lymphoid cells; at a later period numerous pigment cells

make their appearance. In the gray atrophic medulla the basement substance predominates, and the cells which are poor in fat and scanty have an almost epithelioidal character.

According to O. Weber, the bones sometimes contain lactic acid. Their specific gravity is diminished, they are rich in fat, and unusually poor in inorganic constituents, especially the lime salts. In one case, Huppert found phosphate of iron. Not alone simple atrophy and fatty changes, but also degeneration have been found in the muscles.

Many claim that the disease is the result of the formation of lactic acid in the medullary spaces, this giving rise to decalcification of the osseous substance. Rindfleisch attributes a solvent influence to carbonic acid, which is perhaps produced excessively in the medullary spaces as a result of stasis of blood. But Langendorff and Mommsen showed that osteomalacic changes do not consist simply of decalcification. For example, they found, in the basement substance of the bones an imperfect formation of lamellar systems, longitudinal striation and fibrillated formations, and in such places there were often large masses of Sharpey's fibres. Cohnheim does not look upon the limeless places as having been previously healthy, but regards them as an apposition of diseased tissue. The frequent occurrence of the disease during pregnancy has been attributed to the fact that the maternal organism supplies the fœtus with a large amount of lime, and thus retains an insufficient amount for its own consumption.

The obscurities of the problem have not been cleared up by experimentation.

IV. Diagnosis, Prognosis, Treatment.—The disease is distinguished from rachitis by the fact that the bones, instead of remaining soft, grow soft.

The prognosis is unfavorable, and recovery is exceptional. In women there is danger of relapse during pregnancy or of death from difficult labor (narrow pelvis).

The treatment is similar to that of rickets. Busch particularly recommends phosphorus.

7. *Arthritis Deformans. Deforming Inflammation of the Joints.*

I. Etiology.—Arthritis deformans is a disease of advanced age, and is rare before the thirtieth year. It is more frequent in women, particularly among the poor.

The disease is sometimes attributed to heredity, cold, exposure to wet, living in damp rooms, insufficient nourishment, bodily and mental strain. It is observed in women after parturition, too frequent confinements, and excessive lactation. Kohls observed it as the result of fright, and it has also been found associated with diseases of the spinal cord, especially locomotor ataxia. Deforming changes in the joints have also been found in hysteria.

The disease may be the result of injury, such as dislocation, fracture near the joint, contusion, etc. Those joints which are used a great deal are attacked with special frequency, for example, the joints of the fingers in seamstresses, watchmakers, etc.

Gaskoin maintains that arthritis deformans develops after psoriasis, lichen, variola, and particularly after area Celsi.

II. Symptoms.—The disease always develops gradually and slowly. It may attack only one or a number of joints. In the former event, the hip is frequently involved, particularly in old men (malum coxæ senile). Polyarticular arthritis includes two varieties, according as it attacks the trunk and large joints of the limbs, or the phalangeal and

metacarpo-phalangeal joints of the feet and hands. The disease may be strikingly symmetrical.

Obstinate hemicrania has been noticed in women as a prodrome of the disease.

The symptoms generally begin with pains in the joints. These may extend over the entire limb, and are either confined to a definite nerve tract or are of a vague character. Remissions and exacerbations are frequent, the latter occurring particularly in windy, damp, and cold weather. Some patients complain of paræsthesiæ, a feeling of coldness, formication, etc. The joints gradually become stiff, are easily tired, and firm, bony prominences appear, which gradually increase in size. The overlying skin is generally thin, but otherwise unchanged; more rarely it is slightly reddened and inflamed. The longer the condition lasts and the more the ends of the bones are swollen the less movable the joints become. A hard crackling is often felt on moving the joints. The muscles inserted near the joint often undergo rapid atrophy and contracture, thus increasing the deformity.

Fig. 15.

Position of the fingers in arthritis deformans.

In the hands, it is generally found that the second, third, and fourth fingers are flexed in the metacarpo-phalangeal joints towards the ulnar, more rarely towards the radial side, so that the fingers lie across one another like the shingles on a roof (vide Fig. 15). The little finger and thumb are generally unaffected.

In the feet, the great toe is usually affected with the greatest severity.

The spine presents deformities, impaired mobility, and symptoms of compression of the spinal cord and nerves, from narrowing of the natural openings and canals.

If the disease is very extensive, the patients are rendered completely helpless, although life may be maintained for twenty or thirty years. The internal organs are generally intact. Hueter has noticed disease of the endocardium, and early arterio-sclerosis is sometimes noticeable. Death is generally the result of intercurrent diseases.

III. Anatomical Changes.—The capsule of the affected joints first undergoes thickening, and villous proliferation develops upon its inner surface, especially where it folds upon the cartilage. Long, thread-like prolongations extend occasionally into the joint cavity. Some of the villi may undergo ossification, and they may become separated

and float free in the joint. Parts of the capsule itself may be ossified, and sometimes the entire synovial membrane forms a sort of bony capsule.

The ends of the bones are thickened laterally, and proliferate, in a measure, like fungi. Their surface is generally smooth, shining like ivory, and destitute in great part of cartilage. There may be considerable destruction of the osseous substance itself; for example, the neck of the femur occasionally undergoes more or less complete atrophy. As a matter of course, the joint surfaces are thus changed and mobility impaired, and occasionally new joint surfaces are formed.

The tendons inserted into the joint are often thickened, occasionally ossified in places; more rarely they are defibrillated and atrophied. The associated muscles present atrophy, fatty degeneration, and fibrous callosities.

Weichselbaum regards the lesions as the result of advanced, sometimes premature senile changes; others lay stress on its primary inflammatory character. It would appear to us as if there are different anatomical and etiological varieties of arthritis deformans. The process consists essentially of proliferation of the cartilage cells of the articular cartilage, especially at its edge, ossification of the deepest layers, and mechanical atrophy of the middle portions; in addition, gradual defibrillation of the articular cartilage and sclerosis of the underlying bony substance, with progressive proliferation and ossification of the lateral portions of the articular cartilage.

IV. Diagnosis.—The diagnosis is easy. In gout, the great toe is particularly affected, typical gouty attacks occur, and arthritic deposits are found in the cartilage of the ear and in other parts of the body. It may be difficult to distinguish the disease from certain forms of tubercular arthritis, but deformity of the joints is absent in the latter affection. In chronic articular rheumatism, local inflammatory symptoms predominate.

V. Prognosis.—The prognosis is good so far as regards danger to life, but it is difficult to effect permanent recovery.

VI. Treatment.—Among internal remedies, the greatest reliance may be placed on potassium iodide; arsenic and cod-liver oil are also recommended. The joints should be painted with tincture of iodine. During the summer, we may recommend sodium chloride, sulphur, iodine, or mud baths, or indifferent thermal waters.

Good results have been obtained from massage and galvanism (to the sympathetic, spinal cord, nerve plexuses, or the joints).

SECTION X.

INFECTIOUS DISEASES.

A. INFECTIOUS DISEASES WITH TYPICAL LOCALIZATION.

PART I.

ACUTE INFECTIOUS EXANTHEMATA.

1. *Measles. Morbilli.*

I. Etiology.—Measles is an exquisitely contagious disease which is conveyed only by contagion, although, as a matter of course, it must have developed originally in an autochthonous manner.

Experiments have shown that the contagious matter is conveyed in the blood, tears, nasal secretion, sputum, and fluid contents of vesicular eruptions which may form upon the skin. Attempts at infection with scales of epidermis after the subsidence of the eruption are attended generally with negative results, so that the majority of authors deny the infectious character of the disease during the period of desquamation.

The contagious substance possesses the power of leaving the body of the patient and diffusing itself in the immediate vicinity. This is inferred from the fact that the disease may be contracted from mere presence in the sick-room, without contact with the patient or any articles which he has used. To explain the phenomenon, it is assumed that the infectious matter leaves the organism through the exhalations from the skin and lungs.

The virus of measles may also be conveyed through the medium of other individuals and inanimate objects. Hence, physicians may spread the disease from affected families to healthy ones.

It is very important to know that the contagious property of the disease exists during the period of incubation and the prodromal stage. Hence, during an epidemic of measles, all individuals should be quarantined who are apparently suffering merely from simple coryza, cough, and conjunctivitis.

In accordance with the recent views concerning the origin of infectious diseases, we assume that measles are produced by low organisms (bacteria, schizomycetes), but the bacterium has not been discovered with certainty. Babes and Cornil, Braidwood, Murray, and Vacher, and Lebel, have recently claimed to have found the bacteria of measles in the expired air, blood, tears, nasal secretion, and some of the internal organs.

A susceptibility to infection with measles is possessed by the majority of individuals. The greater number are exposed during childhood, so that the disease is regarded as one of childhood. But in isolated places which have long been free from measles, and have been accidentally infected by diseased sailors, it has been found that young and old alike are attacked. A diminished susceptibility is attributed to the first six months of life, although cases are known in which children were attacked with measles a few days after birth, or even at birth. Indeed it is even held that the fœtus in utero may pass through the disease, and therefore remain free from subsequent infection.

Other diseases at the most delay the outbreak of measles. Immunity is not conferred by pregnancy or the puerperal condition. Measles are sometimes associated with another infectious disease. The known combinations are: measles with typhoid fever, variola, scarlatina, erysipelas, roetheln, varicella, pemphigus, and mumps. The combination of measles and pertussis is not at all rare.

As is true of the majority of other infectious diseases, a single attack of measles confers an immunity against subsequent attacks. Cases of double or triple infection are rare. In some epidemics, however, reinfection is relatively frequent. In some cases, many months or years elapse between the two attacks; in others, only a few weeks. Cases in which, a few days after the disappearance of the eruption, another undoubted eruption develops are probably instances of a relapse (extremely rare). Some individuals seem to possess a temporary immunity. Despite opportunity for infection, they escape in one epidemic, but may be attacked in the next.

The disease occurs sporadically, more frequently in epidemics. In large cities, sporadic cases are observed almost constantly. Such cases occasionally form the starting-point for epidemics. Epidemics appear not infrequently at certain definite intervals (two, four, and six years). It appears as if a certain amount of "measles material" must be collected before the disease can spread extensively. The most favorable opportunity for infection is afforded by schools and places of public amusement. The danger of infection is less in the open air than in closed rooms.

Epidemics are more frequent in the winter and spring than in other seasons. Those occurring in the former seasons are often complicated by respiratory diseases, while obstinate diarrhœa is not infrequent in summer epidemics.

An epidemic generally lasts four to six months. As a rule, it rapidly reaches its height, and at this period the cases are generally more serious than at the beginning or end of the epidemic.

At certain times measles spreads pandemically, *i. e.*, over large areas of country. According to Guttcet, the whole of Russia was ravaged, in 1866, by an epidemic of measles.

II. Symptoms and Anatomical Changes.—If the virus of measles is conveyed to a healthy organism, a certain period elapses, during which the poison increases and accumulates in the system before the first signs of poisoning become noticeable. During this stage of incubation, which lasts ten days in typical cases, the individual often feels entirely well.

Although the length of the period of incubation is tolerably constant, nevertheless variations do occur. These will depend, among other things, upon the amount and virulence of the poison introduced, and upon the power of resistance of the individual

This stage is followed by the prodromal period, which lasts, on the average, three days. It is characterized by severe affection of the mucous membranes of the nose, conjunctiva, mouth, pharynx, larynx, trachea, and bronchi. Some writers consider these inflammations as co-ordinate with the subsequent eruption on the skin, and Rehn proposes to call this the stage of mucous membrane exanthem (enanthem).

The third stage is that of the eruption, which appears upon the external integument. In typical cases, it begins on the fourteenth day after infection, and lasts three or four days.

The final stage is that of desquamation, which lasts, on the average, seven days.

During the stage of incubation, especially in the first half, the general health is often undisturbed. In the second half, there are sometimes slight temporary elevations of temperature. The children become irritable and fretful, lose their appetite, suffer from eructations and foul breath, and a coated tongue; sleep is disturbed, or there is unusual somnolence. As the prodromal period approaches, the inflammations of the mucous membrane become noticeable.

The prodromal stage often begins with a single chill or repeated chilly feelings. This is followed by fever, which may reach 40° C. on the first night. On the two following days the temperature may return to the normal, or there is a slight elevation at night, rarely in the morning. Hence many patients feel sick only on the first day of the prodromal stage.

The inflammations of the mucous membranes, which are often observed towards the close of the period of incubation, now increase in severity. The injection of the conjunctiva increases and extends to the conjunctiva bulbi; subconjunctival œdema (chemosis) sometimes develops. The lachrymal caruncle is reddened and swollen so that the tears cannot pass freely into the lachrymal canal, and in part flow over the edge of the conjunctiva. The patients dread the light, complain of itching, burning, and the feeling of a foreign body in the eyes, rub the lids often, and may suffer from spasm of the lids. Nasal catarrh is manifested by impermeability of the nose and a feeling of burning and dryness, which is soon followed by increased secretion. Frequent sneezing occurs, and this may be increased into a sneezing spasm. If the inflammation extends into the frontal sinuses, the patients complain of pain and pressure in the frontal region. Pharyngeal catarrh produces a sensation of dryness and difficulty in deglutition. Buccal catarrh may create a feeling of abnormal heat, burning and dryness in the mouth. Cough, hoarseness, burning in the laryngeal region, and a thickening sensation under the sternum indicate catarrh of the air passages. The cough may become spasmodic, or it grows hoarse and barking, and assumes the sound of the dreaded croup cough.

The objective changes on the mucous membranes are not always alike. In the majority they consist of diffuse redness. Certain parts, for example, the follicles on the palate, often take an active part in the inflammatory swelling, and become visible as small nodules. The injection of the vessels may be unusually marked, and sometimes slight submucous hemorrhages are seen. In other cases, the redness occurs in patches, but these patches may, and in fact generally do, coalesce and produce a diffuse redness.

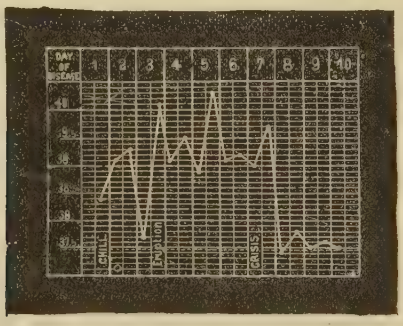
A few autopsies seem to indicate that other mucous membranes may also become inflamed. Patches of congestion have been found upon the

mucous membrane of the bronchi, stomach, intestines, and genitalia, and even upon the pleura. In one case, Weil observed pleurisy.

As the eruptive stage approaches, the bodily temperature suddenly rises to 39° C. or more. As a rule, the temperature continues to rise in the next two days, and at the end of the third or fourth day (usually at night) returns in a crisis to the normal or even subnormal. Then the condition generally remains apyrexial, or there are occasional slight rises of temperature. The height of the fever generally coincides with the greatest development of the eruption.

The eruption on the skin often appears quite suddenly, and this, together with the renewed increase of temperature, sharply separates the prodromal stage from the eruptive stage. In some cases, however, the transition is more gradual. The eruption appears first on the chin, cheeks, and forehead, and soon follows upon the scalp, the integument over the mastoid processes, and the neck. In twelve to thirty-six hours the trunk and limbs are also covered. The patches are most abundant on the face, chest, and back, most scanty on the lower limbs. The

Fig. 16.



Temperature curve in uncomplicated measles.

flexor and extensor aspects of the limb are affected alike, the palms of the hands and soles of the feet are also attacked. Certain parts of the body sometimes escape, or the eruption spreads in a different manner from that described. The occurrence of the eruption is attended occasionally with slight itching.

The exanthem of measles forms red patches, of a round, elongated, or semilunar shape, varying in size from two to six millimetres. On pressure, they grow pale; at a later stage, a yellowish or pale brown patch is left after pressure (combination of exudation and even diapedesis of red blood-globules with the primary hyperæmia). The borders of the patches are sharply defined, and not infrequently irregular and jagged. The patches are slightly elevated. The centre of well-developed patches often forms a prominent papule (provided in many places with a central hair) which is the result of swelling of a sebaceous follicle, and is sometimes better felt with the finger than seen. Both forms are almost always associated with one another. If there has been profuse diaphoresis, the epidermis of the patches may be raised in places into small vesicles. Or the inflammation in the patches may be so active that the blood-vessels rupture and give rise to small cutaneous hemorrhages.

This phenomenon possesses no special significance. The patches may coalesce in places (generally in the face, where it may give rise to œdema of the face and lids), but they never coalesce on all sides.

The eruption extends over the entire body in twenty-four to thirty-six hours, and remains at its height for twelve to twenty-four hours. The patches in the face have occasionally paled before the limbs are attacked. At the height of the eruption we sometimes notice enlargement of the peripheral lymphatic glands and slight enlargement of the spleen. The heart presents systolic febrile murmurs. Diminution of the red and increase of the white blood-globules have been noticed. The tongue has a white coating. Thirst is increased, the appetite is lost. The urine possesses the characteristics of febrile urine; it contains occasionally traces of albumin, often furnishes the acetone reaction, and is said at times to contain sugar (?).

Various statements are made concerning the anatomical changes in the skin. Hebra and Mayr assumed, from analogy, inflammation and swelling of the sebaceous follicles. Simon found the cutaneous glands intact on microscopical examination. The epidermis and cutis were also intact, the latter being swollen into a papule, probably as the result of fluid exudation. Fine molecules, which did not dissolve in acetic acid, were found between the cutis fibres. Neumann recently described dilatation of the vessels of the cutis, emigration of white blood-globules and their accumulation on the outer surface of the blood-vessels, sebaceous and hair follicles, and between the muscle cells of the arrectores pili, and dilatation of the hair follicles at the point of insertion of the arrector.

After the eruption has reached its acme, the patches pale quite rapidly, at first in those parts in which the eruption appeared earliest. The intensity of the color of the eruption occasionally varies slightly, the redness increasing with the increased bodily temperature. The patches often leave yellowish or light-brown pigment spots, which persist into the second week.

The stage of desquamation runs an apyrexial course. It occurs in very fine scales, earliest and most distinctly in the face. It is only slightly indicated on covered, perspiring portions of the body, or if baths have been employed. It is often associated with pruritus. The patient may be regarded as well by the end of the fourth week.

Anomalies in the course of measles are often observed. One of the most frequent is a change in the duration of the individual stages. The individual periods may be longer or shorter, and the eruptive stage is so slight at times that care is requisite in order to recognize the disease. The eruption may also be distributed irregularly and appear in relapses. There are numerous variations in the shape, color, abundance, and distribution of the patches. Sometimes no eruption is observed. The patients were subject to infection, and presented the symptoms of measles, but no exanthem was observed. Desquamation occurs, nevertheless, in some of these cases. In other cases the eruption on the skin was present, but the inflammations of the mucous membrane remained absent.

The disease is sometimes said to run an apyrexial course, but the diagnosis is not always certain in such cases. In others, there is unusually protracted and high fever—a phenomenon which leads us to the consideration of the complications of measles.

The stage of incubation sometimes begins with high fever, followed by somnolence, delirium, and epileptiform convulsions. These symptoms

are not frequent, and their development is favored by accidental intercurrent affections.

The nervous symptoms referred to may also be observed in the subsequent course of measles as the result of abnormally high temperatures. Anatomical diseases of the nervous system are rare, although meningitis has been observed in a number of cases.

A very grave complication is malignant hemorrhagic measles, which is observed with relative frequency in feeble, cachectic individuals. Hemorrhages appear, not alone upon the external integument, but also into the subcutaneous cellular tissue, and from the nose, air passages, gastro-intestinal tract, and genito-urinary apparatus. The temperature is generally high. The patients often lie in a typhoid condition: tongue dry, lips and tongue covered with sordes, abdomen distended, diarrhœa frequent. There is rapid exhaustion and death after symptoms of increasing collapse. This is no doubt the result of a general septic condition.

Every organ may be the site of complications during the course of measles, and some epidemics are characterized by the frequent occurrence of certain complications.

Erythema of the skin is sometimes observed during the prodromal and eruptive stages. Urticaria, more rarely pemphigus, may also be associated with the eruption of measles; herpes facialis is occasionally observed. After the disappearance of the eruption, gangrene of the skin or multiple abscesses and furuncles sometimes develop.

The mucous membranes often present complications, such as phlyctænular inflammation of the conjunctiva, epithelial erosions of the cornea, keratomalacia or ulcerating keratitis, conjunctival blennorrhœa or diphtheria (the latter generally terminates in rapid loss of the eye).

The patients complain not infrequently of impairment of hearing and ringing in the ears, on account of the extension of the catarrh from the naso-pharyngeal space to the Eustachian tube and even the middle ear; the catarrhal inflammation sometimes becomes purulent. If the middle ear contains a large amount of secretion, the patients complain of throbbing and sticking pain in the ear. The membrana tympani not infrequently contains a slit-shaped opening, through which the secretion trickles, but this generally recovers spontaneously. If swelling of the mucous membrane and impairment or abolition of hearing on both sides is left over, deaf-mutism may be produced in young children. Gottstein described desquamative inflammation of the drum membrane.

Epistaxis is frequent, either in the prodromal or eruptive stage, and often affords a feeling of decided relief.

The buccal mucous membrane sometimes contains superficial follicular ulcers or aphthous changes. Sprue is observed in feeble and uncleanly children. Stomacace, gangrene, or noma are rare.

The tonsils are often swollen, occasionally there is phlegmonous amygdalitis, or even diphtheritic and gangrenous changes in the tonsils and pharynx.

Ulcerations have been found on the posterior wall of the larynx, and also on other parts of the laryngeal mucous membrane. A very grave complication is laryngeal diphtheria (croup), which is unusually frequent in some epidemics. Bronchiolitis and broncho-pneumonia are not uncommon; fibrinous pneumonia is observed less frequently. We often find atelectasis and acute pulmonary distention, particularly on the anterior median borders of the lungs. Pulmonary abscess and gangrene and

pleurisy are rare complications. Inflammatory changes in the lungs produce unusually protracted and high fever, and give rise to the danger of suffocation, or, at a later period, of incomplete absorption, tubercular infection, and caseation of the inflammatory products. Complications on the part of the respiratory organs are especially frequent during the winter.

The heart is rarely affected (endocarditis or pericarditis). Demme described enlargement of the thymus gland.

The digestive organs are especially apt to be affected in summer epidemics. The tongue may lose its white coating and, as in scarlatina, become diffusely red and papular from swelling of the papillæ. Frequent vomiting is not uncommon. There is occasionally violent diarrhœa, of a cholera-like or dysenteriform character. I know of one case in which a medical student died in twenty-four hours with choleriform symptoms, shortly after the eruption of measles had paled.

Serious renal changes are rare, although albuminuria, together with the presence of casts, and hæmaturia have been described in a number of cases.

Necrosis and gangrene of the genitals has been occasionally observed.

Complications and sequelæ of measles cannot always be sharply distinguished, since the latter generally develop out of the former. After recovery from measles, previously healthy children sometimes remain weak, and do not regain their former health for a long time. Conditions of blood dissolution sometimes persist for weeks, and are shown by hemorrhages into the skin and mucous membranes, particularly the gums. The skin exhibits a tendency to chronic inflammations, and obstinate eczema, impetigo, and furunculosis, etc., develop. Measles are often the starting-point of scrofula and tuberculosis, enlargement or suppuration of the lymphatic glands, diseases of the joints and bones, chronic pulmonary phthisis, or miliary tuberculosis. Poorly nourished or feeble individuals are especially endangered. These conditions are probably the result of imperfect absorption and drying of the inflammatory products and proliferation of tubercle bacilli, on account of the diminished power of resistance of the organism.

Optic neuritis, chorio-retinitis, and amaurosis have been observed at times as sequelæ; the amaurosis generally recovered in a few days or weeks.

Measles sometimes exert a favorable influence on other diseases, and have been known to cause the disappearance of chronic skin eruptions, epilepsy, chorea, even diseases of the bones and joints. Diseases of the respiratory organs are always rendered worse after measles, and the combination of pertussis and measles is almost always followed by severe pneumonic and bronchitic symptoms.

Uncomplicated measles are rarely fatal, but complications may be attended with great dangers.

III. Diagnosis.—The diagnosis is easy if we direct attention, not alone to the eruption, but also to the remaining symptoms, particularly the fever and changes in the mucous membrane.

Measles are distinguished from scarlatina by the fact that the skin is not uniformly red as in the latter disease, by the absence of the scarlatina tongue and diphtheritic changes in the pharynx, and by the rare occurrence of nephritis. If measles and scarlatina are prevalent at the same time, violent vomiting during the prodromal period would favor the diagnosis of scarlatina.

Roetheln is distinguished from measles by the absence or slight grade of the febrile movement.

If variola is also prevalent, measles may be mistaken for a beginning variola, but in the latter affection papules and pustules very soon develop upon the patches; severe pains in the back during the prodromal stage favor the diagnosis of variola.

It must also be distinguished from roseola due to other causes. Typhus fever has been mistaken for malignant measles. It is easier to exclude typhoid fever, since, in this affection, the roseola is not so profuse. In both cases the face, and generally the limbs are not attacked. Patches of roseola occur occasionally with the menses, after gastric disturbances or the administration of certain drugs, but in such cases there is no fever, and the mucous membranes are not attacked. In syphilitic roseola, other evidences of syphilis are present.

IV. Prognosis.—In uncomplicated measles the mortality is hardly three per cent, but malignant epidemics sometimes occur and are much more fatal. The prognosis is so much more serious the younger the patient, the weaker his constitution, and the more unfavorable the surrounding conditions. High fever, bronchiolitis, broncho-pneumonia, croup, and severe gastro-enteric disturbances also cloud the outlook. Dangerous sequelæ may make their appearance after the subsidence of the measles.

V. Treatment.—Rational prophylaxis may limit the spread of the disease. The patients should be entirely isolated from healthy children, even during the incubation and prodromal periods.

If measles break out in a family, the most certain protection for the healthy members is to send them away to an uninfected place. As the disease can be escaped by very few at some time of life, and as it said to run a more severe course in adults, it has been proposed by some to permit infection if the epidemic is not malignant. Prophylaxis also includes disinfection of the patient's clothing and bedding in hot vapor, thorough airing of the sick-room, and disinfection with sulphur vapor, and the addition of carbolic acid (5%) or corrosive sublimate (1 : 1,000) to the sputum, urine, and fæces. Bath-tubs employed by the patient should be thoroughly scrubbed before being used by others, and their eating utensils should be kept separate.

About one ounce of sulphur should be burnt for each cubic metre space in the room; the sulphur is broken into small pieces, mixed with powdered sulphur, and lighted in a clay vessel. The walls and furniture should first be moistened so that the sulphurous acid may act more effectively. The room should be kept closed at least six hours, and then aired for a few hours.

Uncomplicated measles require no medicinal treatment. The sick-room should be capacious, and aired several times a day through the adjacent room. It should be slightly darkened, and the head of the bed placed towards the window to avoid a glare in the patient's eyes. The temperature of the room should be kept at 15° R. In the winter, open vessels filled with water are placed upon the stove in order to keep the air moist. During the febrile period the patient should receive only fluid food: weak tea, milk, soup, lemonade, carbonated waters, or water mixed with a third claret. The bowels should be kept open daily, a mild laxative being given if necessary. The use of lukewarm baths (26° R.) is extremely important; they should be given between 8 and 9 A.M. and 4 and 5 P.M., duration fifteen minutes. After the bath the body is

quickly dried with warm cloths, and the shirt and bedding are also kept warm. We are convinced that measles run a milder and shorter course in many cases under this plan of treatment with baths.

If the temperature of the body rises above 39.5° C. in the morning, or 40° C. at night, on account of the severe infection or complication with inflammations of internal organs, baths must also be given. We prefer lukewarm baths (for thirty minutes, two or three times a day) to cold baths for this purpose. But if the fever continues unchanged for forty-eight hours, we should order antipyretics. Antipyrin (gr. xxx.–lxv. to ℥ ij. of lukewarm water per rectum) is preferable, on account of its prompt and continued action, to quinine, salicylic acid, kairin, or thallin.

In other respects purely symptomatic treatment must be adopted.

After the fever has ceased for a week, the patients may leave the bed, and at the end of another week may go into the open air if no residua of the disease are left over.

2. *Scarlet Fever. Scarlatina.*

I. Etiology.—Like measles, scarlatina is one of the infectious diseases, and never develops autochthonously. It is often difficult, however, to ascertain the source of infection. Close contact is not necessary to produce infection. It is sufficient to be in the same room with the patient, so that the poison is evidently communicated to the air. Infection may also occur through the agency of persons or objects that have come in contact with scarlatina patients. Two facts must be noted in this connection: First, that very brief contact is often sufficient to produce infection, and secondly, that the virus possesses very great vitality and is capable of producing infection, through the agency of infected objects, at the end of ten years.

The virus is supposed to be present in the blood, lachrymal fluid, nasal secretion, sputum, epidermis scales, urine, and perhaps the fæces of the patient. Cases have been reported of successful inoculations with blood and the contents of miliary vesicles on the skin. But such experiments are not always successful, particularly those performed with scales obtained during the stage of desquamation.

Scarlatina seems to be infectious in every stage, perhaps least in the incubation stage, most during the eruptive stage, next in the stage of desquamation. As a general thing, the patient should be kept secluded until the end of the sixth week.

The nature of the scarlatina virus is not known. Bacteria have been sought for, but with the exception of the recent statements of Pinkus, none point with certainty to the scarlatinous character of these organisms.

The susceptibilty to scarlatina is not so general as that to measles. If measles and scarlatina are rife in any locality at the same time, it is often found that children who had suffered from scarlatina are attacked by measles, while many escape scarlatina entirely. As in the case of measles, some individuals seem to possess a temporary immunity against scarlatina. On the other hand, certain accidental circumstances may create an increased susceptibility. This is true of injuries and recent delivery, especially in primiparæ. Under such circumstances, we must be cautious in making a diagnosis, since erythemata (generally pyæmic or septicæmic) may occur and be mistaken for scarlatina.

It is also said that deaf-mutes possess an increased, phthisical and scrofulous individuals a diminished, susceptibility to the disease.

The majority of individuals suffer from scarlatina during childhood, but more adults are attacked than in the case of measles. Scarlatina is rare in the first six months of life, most frequent from the age of two to seven years. It has been observed, however, a few days after birth, and even at birth. Cases of the latter variety require the strictest criticism, since scarlatina may be mistaken for erythema neonatorum.

In childhood the sexes are affected alike; among adults females are said to be attacked more frequently.

A single attack generally confers permanent immunity, although there are occasional exceptions to this rule. Some individuals have been known to suffer as many as four attacks at intervals of a few years.

Relapses within a few days or weeks after an attack are also rare. The term pseudo-relapses is applied to those cases in which the eruption returns before desquamation has occurred.

Scarlatina is combined occasionally with other infectious diseases, for example, measles, variola, varicella, typhoid fever, and mumps.

Sporadic cases occur constantly in large cities. At times these develop into epidemics, which may spread over a large territory (pandemic). Epidemics of scarlatina run a longer course than those of measles, present repeated exacerbations and remissions, and in isolated cases are often protracted more than a year. In some localities the epidemics are said to appear at certain definite intervals (four to six years). The majority of epidemics begin in the autumn. They are sometimes extremely dangerous.

II. Symptoms.—The disease is divided into the stage of incubation, prodromata, eruption, and desquamation.

The stage of incubation often varies greatly in duration. On the average, it lasts four to seven days. In some it lasts hardly half a day, and in others it is reported to have lasted two or three weeks or even months (?).

The prodromal stage is sometimes barely indicated; in other cases, it lasts twenty-four to forty-eight hours.

During the stage of incubation, the majority of patients feel almost entirely well. In some, there is a general feeling of malaise, and there may be a slight rise of temperature, often only at night, towards the end of this period.

The prodromal stage often begins suddenly. It begins more frequently with repeated chilly sensations than with a single violent chill. The temperature rises very rapidly to 39 or 40°, and even higher. There is a burning feeling in the throat, and often disturbance of deglutition. The movements of the jaws are painful and swollen; painful glands are found behind the angles of the lower jaw. The pharynx is red, and its follicles swollen. At first the redness is sometimes found in patches, which begin at the uvula and then extend to the soft palate and its arches, but do not attack the posterior wall of the pharynx. Gastric symptoms are often prominent, especially repeated vomiting. In children, the high fever sometimes gives rise to delirium and convulsions, which are not necessarily of grave omen.

When the stage of eruption begins, the exanthem appears first on the neck, in the region of the mastoid processes, and on the nape, and then extends over the entire body. It is least distinct in the face, be-

cause it is concealed partly by the flush of fever (thus offering a contrast to the eruption of measles), and the chin, angles of the mouth and nose are apt to be extremely pale (local spasm of the vessels). The back and chest are generally attacked with special severity. The extensor surfaces of the limbs are more affected than the flexor surfaces, with the exception of the dorsal surfaces of the hands and feet. In little children, it is very evident that the scalp is also covered with the eruption. The outbreak of the eruption is sometimes accompanied by slight pricking and itching in the skin.

The exanthem begins in the shape of fine, deep-red or scarlet patches, so that the skin appears speckled. These central patches are surrounded very rapidly by peripheral zones which are less red in color. The patches are so closely aggregated that their peripheral zones unite, and the skin assumes a diffuse red color, in which very fine, dark-red points are visible. The integument is swollen and in places slightly œdematous, so that, for example, the palpebral fissure may be diminished in size, as the result of œdema of the lids. The eruption is sometimes preceded, for a few hours, by temporary erythemata. As a rule, the eruption spreads very rapidly from the neck over the entire trunk (often in twelve to twenty-four hours); sometimes it appears almost at once over the entire body (scarlatina lævigata).

The eruption may present variations in shape. The term scarlatina papulosa is applied to those cases in which the follicles of the skin are markedly swollen, and the patches have a papular appearance. Slighter grades of swelling are observed almost constantly on the forehead and dorsal surfaces of the hands and feet. In scarlatina miliaris, very fine vesicles, with clear, alkaline contents, appear upon the skin. This form is favored by profuse sweating, but may appear independently, and is the result of active exudation between the rete Malpighii and epidermis. The little vesicles sometimes attain considerable size (scarlatina vesiculosa s. pemphigoidea). In scarlatina hæmorrhagica, extravasations of blood occur under the skin. This is a bad prognostic sign, if hemorrhages also occur from the mouth, nose, stomach, intestines, genito-urinary or respiratory organs. In some cases, the eruption of scarlatina appears in circumscribed hyperæmic patches (scarlatina variegata).

The affected skin at first pales completely on pressure. Later, a yellowish or even a dirty, hemorrhagic color is left, showing that simple congestion has been followed by exudation and diapedesis of red blood-globules. Despite the hyperæmia of the skin, the irritability of the walls of the vessels appears to be increased. On stroking the skin with a hard substance, the irritated parts remain pale for some time.

The eruption is most marked on the second or third day. Variations in the intensity of the cutaneous redness occur occasionally and are chiefly dependent on the height of the fever. The redness can also be increased by keeping the body warm.

With the appearance of the eruption, the difficulty in deglutition and the redness of the pharynx increase in severity. The inflammation extends from the pharynx to the mucous membrane of the cheeks and lips, and there produces a burning sensation. Swelling of these parts is absent or very slight; increased secretion is more frequently noticeable. Here and there small extravasations are visible. Many of the follicles are swollen and project as little papules.

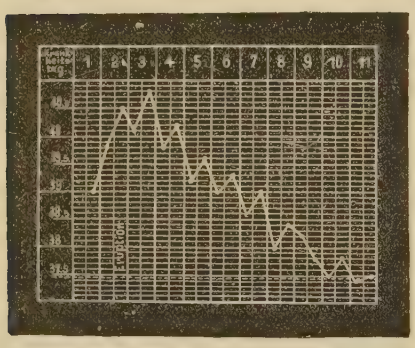
The edges and tip of the tongue are very red, while the larger part of its surface has a more or less thick, grayish, or grayish-yellow coating,

from which the swollen fungiform papillæ project as bright-red papules. In a few days the coating is shed, so that the entire surface of the tongue becomes bright red. The marked swelling of the papillæ makes the surface warty or nodular (strawberry tongue). The size of the organ is generally increased, as is evident from the impressions of the teeth upon its edges.

The bodily temperature rises still higher with the outbreak of the eruption, and, as a rule, exceeds 40° C. The rapidity of the pulse often exceeds one hundred and forty beats a minute. Towards the end of the week the temperature diminishes gradually, not suddenly as in measles. Complications may maintain the elevation of the bodily temperature for weeks.

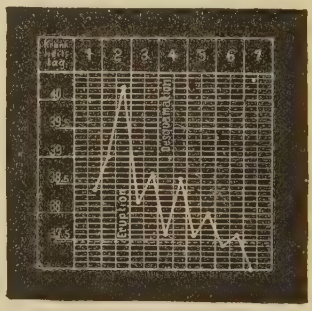
The symptoms mentioned occupy the foreground. Dulness in the head, headache, and delirium are not uncommon. The appetite is lost; thirst is generally increased. Eructations and repeated vomiting are common symptoms. The urine is scanty (febrile urine), often shows

Fig. 17.



Temperature curve in scarlatina of moderate severity.

Fig. 18.



Temperature curve in scarlatina of short duration.

the ferric chloride reaction; Brieger found a large amount of phenol. Febrile systolic murmurs may be heard over the heart. The spleen and even the liver are sometimes slightly swollen. As a rule, the eruption first pales on those parts of the skin which were first attacked, and the stage of eruption is rapidly followed by that of desquamation. Upon the integument of the neck and face, later on the trunk and limbs, appear fissures, and scales of epidermis are raised. Upon the face and sweating portions of the trunk the scales may be small (branny desquamation); on the limbs, particularly the hands and feet, the skin strips off in large shreds, and is sometimes pulled from the fingers like a glove. Small elevations, like vesicles devoid of contents, occasionally form upon the integument, and form the foci of subsequent desquamation. The patients experience the feeling of restored health very soon after the cessation of the fever, and are kept confined to the room with difficulty during the stage of desquamation.

The typical course of scarlatina may be changed by numerous anomalies, complications, and sequelæ.

The individual stages of the disease may vary greatly in duration. The clinical history may consist merely of fever, lasting a few hours,

fleeting redness of the skin, and insignificant disturbances of deglutition. Fig. 18 shows the temperature curve of such a case: sudden repeated vomiting at 12 P.M.; next day, scarlatina eruption over the entire body; the day after, a few remains of the eruption on the limbs; subsequently, marked desquamation. Such mild cases may be followed by severe sequelæ, particularly nephritis. Nephritis in children sometimes appears to be of spontaneous origin, but on careful questioning it is found to have been preceded by brief redness of the skin, followed by desquamation. In other cases, the stages of the disease are unusually prolonged. For example, cases have been reported in which desquamation continued for months. The intensity of the symptoms is also extremely variable, particularly with regard to the febrile movement. Thus other symptoms may be well developed, although the temperature is normal or even subnormal. Again, febrile remissions are sometimes observed at night, exacerbations in the morning. The eruption may also present anomalies. It may appear first on the trunk and limbs, or certain parts of the body remain unaffected. Desquamation often occurs several times on the same place. Or desquamation may also affect, in part, the nails and hair. It is sometimes barely indicated, particularly in old people with a dry, wrinkled integument. Here we may call attention to the fragmentary forms of scarlatina. These include scarlatinous angina without eruption, *i. e.*, a pharyngitis produced by infection with scarlatinous virus, unattended with an eruption, but capable of conveying scarlatina to others. Desquamation is said to occur despite the absence of eruption. In other cases, the eruption develops, but angina is not produced.

Leichtenstern believes that certain forms of nephritis also belong to the category of fragmentary scarlatina. He thinks that during epidemics nephritis may be produced by the action of the scarlatinous virus, without eruption or angina, and that it may give rise, by infection, to fully developed scarlatina. Parotitis, gastro-enteritis, and simple febrile conditions have also been described as fragmentary scarlatina.

The most frequent and important complications are diphtheria, nephritis, and inflammation of the joints.

Diphtheria of the pharynx is almost constant in certain epidemics, and is often more favorable than the primary disease. In some cases, it develops after the outbreak of the eruption; in others, it is present during the prodromal period. It may not produce very severe symptoms at first, so that its onset can only be recognized, in many cases, on inspection. Some cases of scarlatina sine exanthemate appear as diphtheria scarlatinosa sine exanthemate. It is often the starting-point for other dangerous complications. It extends not infrequently to the mucous membrane of the nose. At first, the children present the symptoms of ordinary coryza, complain of stoppage of the nose and a burning sensation. Then a scanty, serous secretion appears, then a stinking, light brownish-red sanguinolent ichor flows almost constantly from the nose, thus proving that the coryza was the forerunner of nasal diphtheria. The nares and upper lip are irritated by the diphtheritic products, erythema and excoriations develop, and the upper lip is very much swollen. In unfavorable cases, there may be destruction of the nasal mucous membrane and necrosis of the bones. The diphtheria may also extend to the Eustachian tube and middle ear, giving rise to tinnitus aurium, impairment of hearing, violent pains in the ear, and usually in-

creased fever. These may eventuate in the production of pus, perforation of the membrana tympani, thrombosis of the sinuses, meningitis, cerebral abscess, uncontrollable hemorrhage, etc. In exceptional cases, the diphtheria extends to the larynx, trachea, and even the bronchi. A more frequent complication is inflammation of the submaxillary lymphatic glands and surrounding cellular tissue of the neck. The parotid and submaxillary glands are sometimes involved. The inferior maxillary region as far back as the mastoid processes is swollen, hard, hot, and painful. At first, the overlying integument is often very pale. If suppuration occurs and the pus is about to perforate the skin, the latter becomes soft, doughy, and reddened. The pus often contains gangrenous shreds of tissue, which is necrosed on account of the compression exercised by the inflamed tissue and the partial interruption of circulation. The pus sometimes makes its way into the mediastinum, pleural or pericardial cavity, or it may produce fatal hemorrhage by erosion of large vessels in the neck.

Opinions differ as to whether scarlatina merely creates a predisposition to diphtheria, or whether the latter is the direct result of the action of the scarlatinous virus on the pharynx. In the latter event, it must be assumed that the diphtheria of scarlatina is essentially different from primary diphtheria. The former view seems better founded, since Loefler found the same micro-organisms in scarlatinous diphtheria as in the primary form. Attention has been called to the fact, however, that the two forms differ clinically, inasmuch as scarlatinous diphtheria rarely extends to the respiratory organs and is still more rarely followed by diphtheritic paralysis. Others claim that there are also anatomical differences, that in scarlatinous diphtheria the membranes are thinner, that necrosed epithelium is situated beneath them, and that fibrinous exudation can be traced deep into the submucosa.

In some epidemics, renal changes are almost constant; in others, they are very rare. Their connection with the height of the fever, intensity of the eruption, and scarlatinous diphtheria cannot always be proven. Nor are they often the result of leaving the bed too early.

The mildest form of renal disturbance is slight transitory albuminuria, which may depend on the fever, the infection, or both. This appears generally in the first days of the disease, and disappears with the fever and eruption.

The condition grows more serious when abundant renal epithelium cells are present in the urinary sediment, an evidence of a desquamative process in the kidneys. In addition, there may be hyaline or epithelial casts in the urine. The latter are sometimes very long, flat, convoluted, and the ends defibrillated (so-called cylindroids). These processes may exist independently or they are associated with albuminuria. In the former event, they may rapidly disappear and form prodromata of an acute nephritis. In some cases, numerous casts have been found, but no epithelium from the tubules. Bacteria have been found repeatedly in the urine, and schizomycetes have also been observed upon the casts.

The symptoms of an acute nephritis often develop quite suddenly. They generally appear from the beginning of the third week to the end of the sixth week, rarely earlier, still more rarely at a later period. The urine becomes scanty and bloody, and contains a large amount of albumin and sediment. Symptoms of uræmia are frequent, and in some cases are the first to direct attention to the urinary changes. Urhidrosis has been observed occasionally in cases of anuria, the urea being deposited upon the skin, after evaporation of the sweat, in the shape of a fine,

white crystalline deposit (vide Vol. II., page 261). In some cases, œdema is the first suspicious sign, and may even appear before the urine contains albumin. It has been found that, despite the existence of nephritis, the urine is temporarily free, in some cases, from albumin, perhaps because certain parts of the kidneys functionate normally, while the secretory power of the diseased portions is entirely abolished. The bodily temperature sometimes rises with the onset of nephritis, and the pulse is often diminished in frequency.

Some writers believe that the kidneys are always affected in scarlatina. While we do not coincide in this view, we admit, nevertheless, that the urine appears normal in some cases, although the autopsy reveals considerable changes in the kidneys.

It is assumed by some authors that the kidneys excrete the scarlatina virus from the organism, and thus undergo inflammatory changes. Others call attention to the relation between diseases of the skin and kidneys, which is seen in other conditions. Still others look upon the nephritis, not as a complication, but as the direct result, like eruption and angina, of the scarlatinous virus.

Inflammations of the joints are much rarer than the renal complications. They sometimes occur at the height of the disease, more frequently after the eruption has disappeared. The small joints of the fingers are especially apt to be attacked, more rarely the large joints of the limbs. The symptoms may consist chiefly of pain, or this may be combined with swelling, redness of the skin, and increased warmth. The phenomena are similar to those of acute articular rheumatism, and, as in the latter disease, the symptoms may jump from one joint to another. The effusion into the joints is generally serous, rarely purulent. The sheaths of the tendons may also undergo inflammation.

Purulent arthritis in scarlatina is sometimes the result of pyæmic conditions. In pus obtained from the joints, Bahrdt and Heubner recently found cocci arranged in chains; these were also found in the diphtheritic deposits on the tonsils, retropharyngeal pus, and in the blood.

We will content ourselves with a brief description of the most important of the remaining possible complications of scarlet fever.

The disease sometimes begins with such a high temperature that the patient is not alone thrown into delirium and convulsions, but dies in a few hours from paralysis of the heart, before the outbreak of the eruption. The temperature may also reach a dangerous height in the further course of the disease, and the patient may then manifest typhoid symptoms (apathy, dry tongue, sordes on the lips, meteorism, diarrhœa). In some cases early death, before the appearance of the eruption, seems to be associated with specially severe infection. Meningitis is one of the rarest complications of the disease. In the majority of cases, the eyes are unaffected. Mild conjunctivitis is occasionally observed. The graver complications are diphtheria, keratitis, keratomalacia, hypopyon, keratitis and iritis, choroiditis, and neuroretinitis. Sudden amaurosis with preserved pupillary reaction, and the changes of retinitis Brightica have been observed occasionally in scarlatinous uræmia. The amaurosis disappeared in a few days or weeks if the uræmic symptoms improved. Auditory disturbances are frequent. Impaired hearing and tinnitus aurium may be the result of occlusion of the Eustachian tube. Graver

conditions are produced by extension of diphtheritic and purulent inflammation into the middle ear.

The pharynx sometimes contains superficial erosions, resulting from destruction of swollen mucous follicles. More serious symptoms are produced by parenchymatous inflammation of the tonsils with abscess formation. The violent pain, high fever, and difficulty in breathing, produced by swelling of the pharyngeal tissues, torture the patient. Danger of suffocation may arise from rupture of the abscess during sleep, and the passage of its contents into the larynx. Gangrene of the pharynx sometimes develops, may spread widely, and prove fatal from exhaustion or the erosion of large vessels. Noma is rarer than in measles.

Laryngeal and tracheo-bronchial catarrh is much less frequent than in measles. Attention has been previously called to the extension of diphtheria from the pharynx to the larynx and deeper air passages. Œdema of the glottis develops occasionally, either as the result of nephritis and uræmia, or of inflammatory changes near the entrance to the larynx. Catarrh and fibrinous pneumonia are rare, pulmonary gangrene and abscess are still rarer.

Inflammations of the serous membranes are not very uncommon; the pleura is attacked most frequently, the peritoneum least frequently. Such inflammations are usually purulent.

Endocarditis is not a rare complication, and the majority of cases of valvular lesion of the heart in children are associated with the endocarditis of scarlatina. It sometimes appears as septic endocarditis, which gives rise to embolic changes in various organs. Hypertrophy of the heart sometimes occurs very rapidly during scarlatinous nephritis. I have also noticed the rapid appearance and disappearance of cardiac dilatation.

Severe complications are sometimes the result of changes in the blood. These result in extravasations beneath the skin and free hemorrhages from various organs, and often prove rapidly fatal.

Obstinate and threatening diarrhœa sometimes occurs, and may assume a dysenteriform character. Hæmoglobinuria and mellituria have been reported in a few cases. Serous, purulent and bloody discharges, abscess, gangrene, diphtheria, and inflammation of the testicle have been described.

Desquamation is sometimes so active that excoriated and bleeding surfaces of skin make their appearance; in rare cases, there is gangrene of the skin or certain parts of the extremities.

The complications of scarlatina cannot be distinguished sharply from the sequelæ, since the latter often develop directly from the former. In some cases, general feebleness persists. The convalescent is weak, even the mental powers are sluggish, and fatal diseases of the respiratory organs or intestinal tract are apt to supervene. Tubercular affections of the lungs, lymphatic glands, bones and joints, rarely of the meninges, sometimes develop after scarlet fever. Some patients suffer from chronic skin eruptions (including furunculosis). Disturbances of audition are frequent after scarlatina. If the disease is bilateral and occurs in the first years of life, it may terminate in deaf-mutism. Extensive destruction of the petrous portion of the temporal bone results occasionally in facial paralysis. Foerster reported a case in which scarlatina had given rise to bilateral deafness and facial paralysis, and, associated with the latter, bilateral ulceration of the cornea and blindness. Accommodative asthenopia is sometimes observed after scarlatina.

Like other infectious diseases, it may also give rise to paralyses. Chorea has also been observed. Suppuration of the joints may result in ankylosis. Valvular lesions of the heart are relatively frequent sequelæ. The renal changes generally disappear in cases which do not terminate fatally; chronic nephritis is a rare outcome, but I have seen two cases of this kind. Zinn described a case of melituria which lasted a long time. Wollenberg observed albinism of the skin and hair after very marked desquamation.

In rare cases, scarlatina exercises a favorable influence on existing diseases. Thus, Gibney observed spontaneous recovery of an obstinate coxitis after scarlatina, and Thompson reports that chorea disappeared in two cases.

III. Anatomical Changes.—In the corpse nothing remains visible of the eruption, but the skin is often peculiarly tense and œdematous.

Microscopical examination of the skin shows swelling of the cutis tissue, ampullary dilatation of the vessels, swelling of the cells in the rete Malpighii, especially of the nuclei; in the deeper layers of the rete are spindle-shaped, elongated cells, and between them an accumulation of round cells and red blood-globules, and an accumulation of round cells around the excretory ducts of the follicles. Fenwick described hemorrhages into the sweat glands, and active desquamation of their epithelium.

The muscular tissue is not infrequently very pale and brittle; the microscope shows cloudy swelling and fatty degeneration of the muscular fibres.

There is not infrequently swelling of the entire lymphatic gland system: the peripheral and mesenteric glands, the solitary follicles, and Peyer's patches in the intestines. E. Wagner has described lymphoid new-formations in the liver, spleen, kidneys, and intestinal mucous membrane. The enlarged follicles of the intestines sometimes undergo ulceration. Klein found hyperplasia of the lymph follicles of the root of the tongue, the pharynx, tonsils, larynx, and trachea; the mononuclear lymph cells were scanty, the multinuclear cells very numerous, so that giant cells were abundant. The veins of the cervical glands were occluded by plugs of fibrin. In the spleen, the microscope showed thickening of the sheaths of the arteries, hyperplasia of the muscle nuclei in the arterial walls, hyaline swelling of the intima, progressing to occlusion; changes in the Malpighian bodies similar to those in the peripheral glands.

The heart is often very flaccid, pale, and yellowish in color, and presents dilatation and hypertrophy. The muscular fibres are often in a condition of fatty degeneration and cloudy swelling. The blood is dark and its coagulability diminished; the white globules are not infrequently increased.

Diphtheritic changes are sometimes found in the intestines, more rarely in the œsophagus and stomach, to which they have probably spread from the pharynx. In hemorrhagic scarlatina, the intestines sometimes contain bloody masses. The liver often presents cloudy swelling and fatty degeneration, with proliferation of round cells in the interstitial connective tissue. Harley states that the solid constituents of the bile are diminished; the biliary acids are sometimes entirely absent.

The kidneys are generally enlarged. In recent cases, extravasations

are visible upon their surface and upon cut sections; in older ones, the organ has a yellowish color, produced by fatty processes.

Friedlaender describes three forms of scarlatina kidney, which are rarely associated and never pass into one another. *a*. Initial catarrhal nephritis; this appears with the eruption, or a few days later, soon disappears, and is characterized by cloudiness, swelling, and desquamation of the epithelium of the renal tubules; a few round cells in the interstitial tissue. *b*. Glomerulo-nephritis; this form is almost characteristic of scarlatina. The glomeruli are enlarged, the nuclei of their walls increased in number, the walls of the coils of vessels thickened, the epithelium of the capsule thickened, sometimes proliferated. *c*. Large, flabby hemorrhagic kidney (septic interstitial nephritis). This depends less upon scarlatina than upon complicating diphtheria and inflammation of the cellular tissue of the neck, and generally proves fatal in a short time. The kidney is large, flabby, infiltrated with large and small hemorrhages, with numerous round cells in the interstitial tissue; micrococci emboli are frequent. Deposits of lime salts in the tubules have been described.

IV. Diagnosis.—The recognition of scarlatina is easy, if symptoms other than the eruption are also taken into consideration. Primary diphtheria of the pharynx is sometimes associated with erythematous cutaneous changes; but these disappear rapidly, as a rule, and are not followed by desquamation. A similar eruption has been observed in acute articular rheumatism. The diagnosis from measles, rubeola, and medicinal eruptions is made according to the rules laid down on page 105.

V. Prognosis.—The prognosis is always serious. Cases which have run a mild course may prove fatal by complications and sequelæ. Death sometimes occurs at the onset of the disease, on account of the high fever and severe infection, and not infrequently quite suddenly. In some epidemics, death is exceptional; in others, it is the rule. As a general thing, the prognosis is so much more serious the younger the patient. The greater the number of complications the less favorable the outlook. Uræmic symptoms, purulent inflammations of the serous membranes, and septic endocarditis render the disease especially grave.

VI. Treatment.—The treatment is similar to that of measles. Prophylaxis is extremely important. The patients must be strictly isolated, and all articles used by them thoroughly disinfected, in order to prevent the spread of the disease. The patients may not be allowed to enter into general communication with others until at least a week after every trace of desquamation has disappeared. If possible, the other children in the family should also be isolated. As a rule, the physician should visit his scarlatina patients last, and change his clothing on visiting patients who do not suffer from the disease.

After scarlatina has developed, it requires the same treatment as measles; we can especially recommend the use of lukewarm baths. Otherwise, the treatment is purely symptomatic.

3. *Roetheln. Rubeola.*

I. Etiology.—Some maintain that rubeola is a special form of measles; others, that it is a mild and peculiar scarlatina; finally that it is a simple roseola. In our opinion, it is undoubtedly an independent infectious disease. It is most frequent in childhood, and rarely occurs in adult life. Nurslings generally escape.

The disease is contagious and is generally acquired by contact with

other patients, or by remaining in the same room; but it can also be conveyed indirectly by other persons or objects. Slight contact is sometimes sufficient.

One attack almost always confers immunity against another. Relapses are rare. The disease does not afford immunity against measles or scarlatina—an evidence of its distinct character.

In large cities roetheln is often sporadic, and at certain intervals epidemics break out (generally during the first half of the year). Their duration varies. Schools and overcrowded houses furnish the most favorable localities for the spread of the disease, which is probably contagious in all stages.

II. Symptoms.—The period of incubation varies from two and one-half to three weeks.

Prodromata are sometimes entirely wanting. In other cases, the patients complain of malaise and anorexia for one to three days. Slight fever (38 to 39° C.) may develop, the patients complain of slight difficulty in deglutition, cough and sneeze a good deal, complain of slight epiphora and photophobia. A few hours later the eruption appears.

But as we have remarked above, prodromata may be entirely absent, and the symptoms mentioned then appear with the eruption. This consists of pale-red, roseolar patches, from the size of a pin's head to that of a bean, which are slightly elevated, and grow pale on pressure. The patches are generally round, with indistinct borders, and often send prolongations into adjacent patches. In places they coalesce. Emminghaus observed erythema as a prodrome of the eruption. In rare cases a few miliary vesicles and petechiæ are noticed.

The eruption appears first on the face and scalp, and then extends to the trunk and limbs. It has generally paled in the face by the time that the lower parts of the body are affected, since the eruption only lasts a few hours.

Some patients complain of slight itching of the skin. The turgor of the skin increases, and there may be slight œdema of the face. The peripheral glands are often swollen, particularly the cervical and auricular glands.

The eruption is sometimes followed by slight desquamation.

The outbreak of the eruption is constantly accompanied by slight catarrhal inflammation of the mucous membrane of the pharynx, respiratory organs, and conjunctiva. The pharyngitis often appears in patches, particularly upon the middle of the uvula. It never attains the severity of the pharyngitis of scarlatina, and disappears as the eruption pales (one to three days).

The temperature may remain unchanged; a slight rise is often noticed at the outbreak of the eruption.

The general condition is often entirely undisturbed. Albuminuria has been observed in a few cases, and œdema of the subcutaneous cellular tissue and tonsillar hypertrophy as sequelæ.

III. Diagnosis, Prognosis, Treatment.—During epidemics, the diagnosis is easy. In sporadic cases the differentiation from measles, scarlatina, and roseola is not always possible.

The prognosis is good; a fatal termination is exceptional.

Treatment is purely dietetic and symptomatic.

4. *Typhus Fever. Exanthematic Typhus.*

(*Spotted Fever, Petechial Fever.*)

I. Etiology.—The mode of infection in typhus fever is the same as in measles and scarlatina, *i. e.*, by personal intercourse and contact. The more intimate and prolonged the contact the greater the danger of infection. Hence, in hospitals, nurses are affected most frequently, next the house-staff, and lastly the visiting physicians.

The patients must be kept in separate wards, since the disease is sometimes conveyed to adjacent beds, and thence to other wards. When the patients are quarantined, the danger of infection is so much greater the larger the number of patients, the smaller the ward, and the less it is ventilated. Further infection is sometimes prevented by keeping the doors and windows open.

The infectious matter adheres not alone to the person of the patient, but also to his clothing and other articles of use. In hospitals, those nurses are often attacked upon whom devolved the duty of storing away and disinfecting the clothing.

Intermediate persons may also spread the disease. Some individuals possess a temporary or permanent immunity, but nevertheless may carry the infectious matter in their clothing, and thus convey the disease to healthy individuals.

In all probability, the virus is contained in the exhalations from the skin and lungs. Experience seems to show that the disease is infectious in all stages, and perhaps during the first part of convalescence.

The nature of the virus is entirely unknown. No schizomycetes have been found in the blood, and attempts to convey the disease to animals have always been attended with negative results.

Typhus fever is endemic in certain regions. Ireland is, in a measure, the classical home of the disease, and it often follows Irish emigrants to England, Scotland, and America. The European continent also contains abiding places of typhus, for example, certain parts of Russia, Galicia, Hungary, and Italy. It is a disease par excellence of the lower classes, so that foreign workmen and tramps must be regarded as its propagators. It has often been found that workmen on railroads, etc., who have come from typhus districts, have spread the disease in their new home.

It has been ascertained, in a number of instances, that large cities are converted artificially into endemic sites of typhus fever. Thus, Berlin always contains a few cases of the disease, which are derived from filthy, crowded lodging-houses. A tramp who leaves such a lodging-house may be attacked by the disease two or three weeks later in some remote locality, and this explains the fact that many cases appear to develop autochthonously.

In our opinion, the disease never arises in an autochthonous manner. If we assume that it is the result of specific lower organisms, this in itself excludes an autochthonous development. Such a mode of genesis is supposed to be proven by the fact that the disease occurs in epidemics during times of war and famine, in crowded prisons, ships, and hospitals, which were supposed to favor processes of decomposition. It has been repeatedly shown that this interpretation is false. Thus, it was demon-

strated, with regard to an epidemic in East Prussia, that the failure of the crops corresponded merely in point of time with the epidemic spread of the disease, and that this had been preceded by the appearance of a few cases of typhus which had been imported by workmen from Upper Silesia.

The disease is mainly one of personal intercourse, so that climatic and telluric influences are not noticeable. Nor is any influence exerted by the character of the soil, the height of the locality above the sea, and the character of the water. As a general thing, seasons exert no influence, although epidemics are somewhat more frequent in winter and spring.

Men are attacked somewhat more frequently than women, probably because they are more exposed to infection. In some epidemics, however, more women were attacked than men. It is said also that the female sex predominates in childhood.

The largest number of cases occur between the ages of 15 and 25 years. The disease is rare before the age of five years, and only a single case has been observed in the first year of life. It grows infrequent beyond the age of 45 years, although cases occur even so late as the age of eighty.

The lower classes are the chief sufferers, but the disease may also be conveyed accidentally to the higher classes. In the latter event it is said, as a general thing, to run an unfavorable course.

Privations, grief, worry, and excesses of all kinds probably increase the susceptibility to the disease by rendering the organism more accessible to the virus.

As a rule, only a single attack is experienced, but a few cases have been reported in which individuals were attacked two or three times. One or more relapses have also been observed, *i. e.*, the symptoms were renewed a few days after the subsidence of the fever.

Epidemics of typhus fever are often associated with typhoid and relapsing fever, since the conditions which favor the spread of typhus act in like manner with regard to typhoid and relapsing fever. In some instances an epidemic of typhoid fever diminished in a striking manner, while typhus fever began to spread. In a number of cases, the patient suffered first from typhoid fever, and immediately afterward from typhus fever. Niemeyer observed the coincidence of intermittent and typhus fever, and the combination of small-pox and typhus has also been described. The latter has also been known to develop immediately after scarlatina.

II. Anatomical Changes.—Typhus fever presents no specific anatomical changes. In general the appearances are merely those of an acute infectious disease.

Rigor mortis lasts but a short time, and decomposition begins rapidly.

In addition to livores mortis, the skin sometimes contains indistinct bluish-red patches and petechiæ. The lips, gums, tongue, and nose are covered with sordes. The body appears well nourished. The muscles are dry, dark-red, of the color of ham. Neumann found Zenker's degeneration of the muscular fibres, in addition to granular and fatty degeneration. The muscles may contain hemorrhages, and the rectus abdominis may be the site of hemorrhagic inflammation, as in typhoid fever.

Similar changes occur in the heart muscles. The blood is generally

dark red, and exhibits little tendency to coagulation. There are evidences of bronchitis, atelectasis, hypostasis, catarrhal and fibrinous inflammation of the lungs. Larsen also observed small pulmonary hemorrhages.

The gastro-intestinal mucous membrane is not infrequently swollen and hyperæmic. Virchow found fissures in the gastric mucous membrane which had given rise to hemorrhages. The solitary and agminated follicles of the intestines are not infrequently slightly swollen; whether they ever undergo superficial ulceration appears to us to be doubtful; at all events, this occurs only in exceptional cases. The mesenteric glands may also be swollen and hyperæmic. The spleen is generally very large, dark red, and soft, sometimes almost diffluent. In Salomon's case it weighed twenty-three ounces (nearly three times the normal). It occasionally contains wedge-shaped and simple hemorrhagic infarctions, sometimes small abscesses as in relapsing fever (?).

The liver is usually enlarged, its cells are in a condition of cloudy swelling and fatty degeneration; nuclear proliferation is noticeable in the intralobular and interlobular connective tissue. Similar changes are found in the kidneys.

Menigeal and cerebral hemorrhages and œdema may be found in the brain. Popoff noticed infiltration of the ganglion cells with round cells, round cells in the periganglionic spaces between the nerve fibres and in the adventitious lymph sheaths; also pigment infiltration of the ganglion cells. Similar appearances are also found in typhoid fever. Small multiple lymphomata were found in two cases.

Beveridge recently observed swelling of the ganglia of the cervical sympathetic.

III. Symptoms.—The stage of incubation has a variable duration. In some cases it is said to have lasted only a few hours, in others as long as one to three weeks. According to Naunyn, it is shorter in children than in adults.

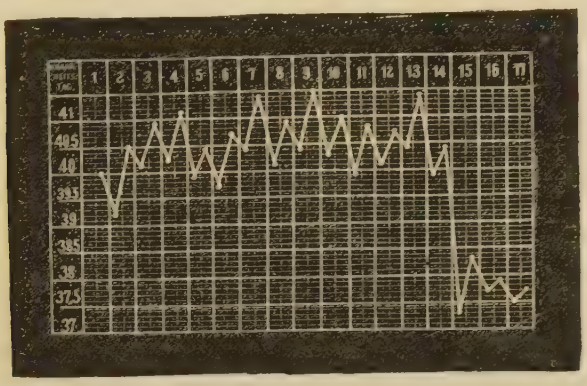
As a rule, the prodromata begins with a single chill, or repeated chilly sensations. In some patients, there is frequent vomiting, others complain of anxiety and oppression in the epigastric region; children may suffer from eclampsia. The temperature rapidly rises, and very soon reaches 40°, 41° or even more. At the same time the pulse becomes hard, and its rapidity may exceed 100 beats a minute. The patient grows so weak and dizzy that he soon takes to bed. In a short time unconsciousness supervenes, and very soon afterwards delirium develops. The patients complain of ringing in the ears and impairment of hearing. The face is reddened and turgid, the eyes fixed and glassy, the conjunctiva injected. The tongue has a gray or grayish-yellow coating, and, like the lips, soon become sticky, dry, and fissured. They bleed readily, the blood dries, and sordes form. The patient suffers from unquenchable thirst, while the appetite is almost entirely lost. The liver and spleen are generally tender on pressure, the latter organ enlarges very rapidly. At the end of a few days the urine often contains albumin. The bowels are generally constipated, later slight diarrhœa may occur.

The prodromata are often preceded for one or two days by vague general symptoms, such as malaise, a feeling of dulness in the head, want of desire for mental and physical activity, loss of appetite, etc.

After the prodromal stage has lasted three to five days, it is followed by the stage of eruption.

The eruption appears first on the breast and abdomen, and then extends to the trunk and limbs. The face does not escape, but in many cases the eruption is here distinctly visible only in individuals with a delicate, pale skin (particularly children). It is often very profuse upon the limbs, particularly on the extensor surface of the forearm. The eruption consists of round, pale-red patches (roseola), which at first grow entirely pale on pressure. After two to four days, the redness changes to a livid color, the boundaries of the patches become indistinct, and yellowish or brownish spots are left after pressure. The primary hyperæmia has evidently been followed by exudation of serum and diapedesis of red blood-globules. The patches often number several thousands. They generally lasts ten days or longer, *i. e.*, into the period of convalescence. Their disappearance is followed by very fine desquamation, rarely by desquamation in larger patches.

Fig. 19.



Temperature curve in moderately severe typhus fever, running a regular course.

In some cases, the roseola presents almost the shape of acuminated papules; small vesicles develop occasionally in their centre.

Petechiæ and vibices sometimes appear upon and near them. Drasche observed herpes labialis in two cases. Miliaria appears as the result of sweating, particularly at the crisis.

The bodily temperature is important in diagnosis. It does not rise gradually, as in typhoid fever, but suddenly, and also falls rapidly and critically to the normal. The fever is continued, and not infrequently rises to 40° or 41°. A slight remission sometimes occurs towards the end of the first week, but at the beginning of the second week the temperature generally rises again (sometimes higher than before) and, as a rule, sinks in a crisis to the normal on the fourteenth to the seventeenth days. The crisis sometimes occurs as early as the eighth or tenth day, sometimes as late as the twenty-first day, or even later.

The crisis, which is attended with profuse diaphoresis, often terminates within twelve hours. In other cases it occupies two or three days (protracted crisis), and sometimes defervescence occurs almost in a lysis. The onset of the crisis may be preceded for a few hours by enormous rise

of temperature, chill, vomiting, and other threatening symptoms, but the hoped-for symptoms of the crisis soon make their appearance. Pseudo-crises are also observed, *i. e.*, the temperature falls at the ordinary period, but rises to its former height on the following night, and then the definitive fall of temperature takes place. Immediately after the crisis, the temperature is not infrequently subnormal.

During the crisis, the majority of the patients fall into a deep, refreshing sleep from which they awake with a feeling of relief. In not a few cases, however, it is followed by such profound exhaustion that death ensues. After the crisis, the urine contains an abundant sediment of urates.

The temperature sometimes rises very rapidly for a few hours before death.

The pulse is generally increased in frequency (100 to 120), corresponding to the temperature of the body. If its rapidity exceeds 120 a minute the prognosis is very grave. The pulse is sometimes irregular and slow; dicrotism is much rarer than in typhoid fever. After the crisis, the pulse is generally normal, occasionally subnormal.

Consciousness is almost always disturbed, partly as the result of the high fever, partly on account of the infection. At first there is violent headache and occasionally well-developed neuralgia, but soon the sensorium becomes clouded; some of the patients lie quietly in a dreamy state, others are violently delirious. The bowels are often evacuated involuntarily, or the patients experience no desire to urinate, and the bladder is distended to the umbilicus. Tremor of the tongue and face, hesitancy in speech, picking at the bed-clothes, and subsultus tendinum are not infrequent.

The lips, tongue, and nares are generally dry and fissured, bleed, and become covered with sordes. Mosler found that the parotid saliva, discharged through a canula, was acid; he recommends catheterization of Steno's duct to prevent parotitis which, in his opinion, is often produced by occlusion of the duct with secretion. Conjunctival, nasal, and pharyngeal catarrh are noticeable, and if consciousness is sufficiently clear, the patients complain of photophobia, dryness and burning in the nose and pharynx. The pupils are generally narrow; according to Schneider, often unequal. There is generally impairment of hearing and ringing in the ears, partly from tubal catarrh, partly from catarrh of the middle ear and inflammation of the drum membrane.

The thorax almost always presents signs of dry bronchitis. The heart is not infrequently dilated towards the right. Occasionally, we may hear febrile systolic murmurs; in severe cases, the first sound may be inaudible (weakness of the heart).

The spleen and liver, particularly the former, are enlarged and tender on pressure. Slight meteorism and tenderness in the epigastric region are observed in rare cases. Nausea, singultus, and vomiting are also rare.

The urine presents the characteristics of febrile urine: small quantity, dark-red color, very acid reaction, high specific gravity, increased amount of urea, uric acid, and kreatinin, diminution of chlorides. Unruh found that the amount of urea was considerable shortly before the crisis, fell on the day of the crisis, and became unusually high two or three days later. Moderate albuminuria is frequent. Griesinger found casts and epithelium of the tubules and bladder in the sediment. Frerichs found leucin and tyrosin.

Thirst is always increased, and even apathetic patients will eagerly drink water. The appetite is lost, the bowels are generally constipated.

The skin is said to have a peculiar mouldy smell. It is generally dry and hot; in rare cases, there is diaphoresis apart from the crisis.

Death may occur from excessive rise of temperature before the outbreak of the eruption, or it takes place at the height of the disease, or shortly before or after the crisis. It is generally the result of heart failure.

Among the anomalies may be mentioned typhus without eruption; also abortive cases of short duration. In other cases, finally, the symptoms are very mild and brief.

Complications and sequelæ are not uncommon. Eclamptic attacks are sometimes observed at the height of the disease. Purulent meningitis was recently described by Huguenin, who also observed embolic changes in the brain. Impaired memory and imbecility sometimes persist for a long time after the disease. Paraplegiæ have been observed, apparently of myelitic origin. Radial neuritis has been described, and it is also possible that purely myopathic paralyses may develop. Clonic twitchings, aphasia, and neuralgias have been reported as sequelæ.

Chronic purulent inflammations of the ear and deafness may occur; the former may extend to the cerebral meninges. Amaurosis has also been mentioned in a few cases.

Moers observed hemorrhages from catarrhal ulcers of the pharynx. Epistaxis occurs occasionally. Hæmoptysis set in in one case, although no pulmonary abnormalities could be discovered. Wojciechowski described hæmatemesis. Intestinal hemorrhages are rare (six times in seven thousand cases, according to Murchison). Necrotic and diphtheritic changes in the intestinal mucous membrane sometimes cause dysenteriform evacuations. Peritonitis is a very rare complication. Murchison described acute yellow atrophy of the liver in one case; Horn reported rupture of the spleen. Cystitis and pyelitis are rare. Diphtheria of the pharynx and larynx has been observed in a few cases, also laryngeal ulcerations. Hypostasis, inflammation, embolism, abscess, gangrene, and miliary tuberculosis of the lungs have been described; likewise pleurisy, pericarditis, and endocarditis. Degeneration of the heart muscle and death from heart failure are not uncommon. In such cases, the extremities often become cool and cyanotic, while the interior of the body retains its elevated temperature.

Embolism and marantic thrombosis may occur in the peripheral veins and arteries. Bed sores may develop, despite the utmost care. Among the complications we may also mention furunculosis, multiple abscesses, erysipelas, parotitis, pyæmia, septicæmia, suppuration of the lymphatic glands, noma, or gangrene of the skin.

IV. Diagnosis.—The disease is differentiated from typhoid fever by its sudden onset and termination in a crisis, and by the fact that the eruption is more profuse, especially upon the face and limbs. Furthermore, diarrhœa, ileo-cæcal gurgling and pain are rare in typhus. It is distinguished from measles by the fact that the latter generally attacks children, and that conjunctival, nasal, and pharyngeal catarrh is very prominent.

V. Prognosis.—The disease is always very grave, although the prognosis depends mainly on the character of the epidemic (the mortality varies from five per cent to more than sixty per cent). The prognosis is so much more grave the higher the fever, the more rapid the

pulse, the weaker the constitution, the more advanced the age, and the severer the complications. As a general thing, a profuse eruption indicates a severe course. The vigor of the heart's action must be taken specially into consideration in regard to prognosis.

VI. Treatment.—The spread of typhus can only be prevented by the strictest quarantine.

If typhus has appeared in a lodging-house, prison, etc., the place should be closed, all its utensils which possess only slight value should be burned, more expensive ones thoroughly disinfected, and the rooms disinfected and aired for a long time.

The patients should be strictly quarantined, and have their own nurses, utensils, and physicians. The latter should visit their typhus patients last, then carefully disinfect themselves, and change their clothing. The patients may receive no visits, and not enter into communication with the outside world. Burial should be strictly private.

Alcohol in large doses (brandy, wine, champagne) or other stimulants, and antipyretics must be administered. Among the latter, we prefer antipyrin (ʒ i.–iss. by enema). Treatment with cold baths produces very little good effect.

In other respects, the treatment is purely symptomatic.

The rules concerning nursing and diet, which will be laid down in discussing typhoid fever, also hold good.

5. *Erysipelas.*

I. Etiology.—Erysipelas may develop whenever, after a wound of the skin or mucous membrane, certain bacteria (erysipelas cocci) gain access to the lymph vessels, and set up a specific inflammation of the skin. The disease belongs, therefore, to the domain of surgery rather than to that of internal medicine.

Until recently, it has been held that erysipelas sometimes develops spontaneously. It will be granted by every physician that cases sometimes occur in which a wound cannot be discovered, but it is also true that the more thorough the search the more frequently will lesions be found, so that we must ask ourselves whether, in the former event, the primary lesion may not have healed and escaped discovery.

Some writers speak not alone of erysipelas of the skin, mucous or serous membranes, but also of erysipelas of the viscera, for example, the lungs (vide Vol. I., page 303).

Erysipelas sometimes appears in epidemics, particularly in the spring. They may extend over an entire city or only to a few houses.

It is sometimes found that operations are followed by erysipelas when new wards are opened. In some hospitals, certain rooms and beds are notorious as the abiding places of erysipelas.

The introduction of a single case into a hospital may lead to its further spread. In other cases, its frequent occurrence is connected with overcrowding and bad ventilation of the wards. Koening described an epidemic which took its start from the operating table, inasmuch as the pillow had been saturated with blood and secretions from a patient suffering from erysipelas, and had not been changed in a long time. Instruments and bandages may also serve to convey the disease.

It is generally assumed, but not positively proven, that the virus is conveyed only a slight distance in the air; infection takes place very

readily through the agency of intermediate persons and inanimate objects. During an epidemic, a very trifling wound (leech bite, hypodermic injections, etc.) may be followed by erysipelas.

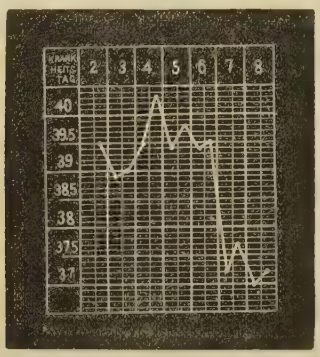
Erysipelas very often follows chronic inflammations and eczemas of the nasal mucous membrane, and is apt to relapse a number of times in the course of a few months or years. Inflammations of the lachrymal sac and duct are a frequent starting-point of the disease. It is sometimes associated with a gum-boil, eczema of the external ear and auditory canal or other parts of the body, and ulcers of the leg.

It occurs occasionally as a puerperal complication, starting from the uterus, and, in the new-born, may follow tying of the cord.

Vaccination erysipelas is a special form which will be discussed later.

Erysipelas is one of those infectious diseases which, instead of conferring immunity against a subsequent attack, produces an increased susceptibility. It sometimes complicates typhoid and typhus fever, relapsing fever, intermittent fever, diphtheria, dysentery, pneumonia, etc. Occa-

FIG. 20.



Temperature curve in a moderately severe case of facial erysipelas.

sionally it exercises a favorable influence on other diseases, for example, syphilis. Erysipelas has been known to be followed by the disappearance or diminution in size of cancer, fibroma, nævi, tumors of the lymphatic glands, phagedænic ulcers, elephantiasis, and lupus. Kopff recently described a case of acute articular rheumatism which rapidly recovered after an outbreak of erysipelas.

A number of successful inoculations of animals have been made, partly with the contents of vesicles, partly with blood. Fehleisen recently inoculated human beings with artificial cultures of erysipelas cocci. He found that if a second inoculation was made soon after a successful one, it was attended with negative results and only proved successful after a certain lapse of time. Jaenisch and Neisser observed a fatal result after inoculation, with erysipelas cocci, of a woman suffering from cancer of the breast.

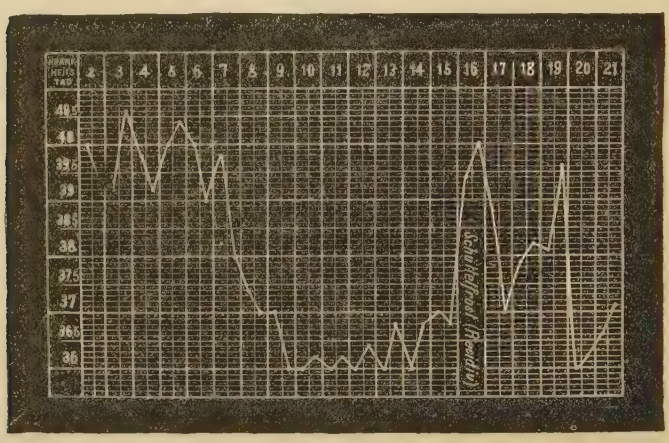
II. SYMPTOMS.—The duration of the period of incubation varies from one to eight days. Heiberg claims that, during an epidemic in Rostock, he observed an elevation of temperature two hours after making a surgical incision.

Prodromata are often wanting. Some patients complain of malaise, anorexia, and pains in the limbs.

The symptoms often begin with a chill, or repeated chilly sensations. This is followed by fever, which may reach 40° C. or more in a few hours. This generally runs a continued course so long as the cutaneous changes last, and usually terminates in a crisis (vide Fig. 20). The occurrence of the crisis is sometimes preceded by sudden, marked elevation of temperature, delirium, chill, etc. If the disease terminates unfavorably, the temperature not infrequently rises very high before death and sometimes even for a little while after death. Relapses may be accompanied by another rise of the normal temperature (vide Fig. 21). In a few cases mild erysipelas runs an apyrexial course. The elevation of temperature is attended with increased frequency of the pulse, anorexia, and increased thirst.

In cutaneous erysipelas, the patients complain of pricking, itching, and pain in the affected parts. The skin is œdematous, devoid of folds and shining (inflammatory œdema), and is hot and red. The epidermis

Fig. 21.



Temperature curve in a moderately severe case of facial erysipelas, with a relapse on the sixteenth day.

is often, though not always, raised in larger or smaller vesicles, whose contents are at first serous, later cloudy, or even purulent. Some writers claim that vesicular elevations of the epidermis are always visible with the aid of a magnifying glass. Excessive tension of the skin may be followed by gangrene. The skin then assumes a blackish-red or greenish-black appearance, the vesicles become filled with hemorrhagic contents and rupture, resulting in necrosis of the parts.

Erysipelas always exhibits a tendency to spread, and in some cases may finally extend over the entire body. The extension occurs along the folds and furrows of the skin. A sallow, yellow œdematous zone first forms, and is followed by hyperæmia of the part.

Red stripes, which correspond to inflamed cutaneous lymphatics, very often start from the periphery of the erysipelatous patch. The adjacent lymphatic glands are generally swollen and painful.

Upon non-hairy integument the extension of erysipelas is directly visible ; it may be assumed to be taking place on the scalp when the lat-

ter becomes painful, swollen, and hot. Upon separating the hairs, redness is sometimes visible. Marked swelling of the skin often produces great deformity. For example, the eyelids may be swollen to such an extent that the patient is unable to open them; the nostrils are not infrequently narrowed and impermeable; and the ears and lips may be converted into shapeless masses.

As a rule, the erysipelatous changes reach their highest development on the third day after their appearance. The redness and swelling then diminish; finally desquamation takes place. The vesicles which have been present dry into thin scales and crusts.

At the height of the disease, the fever and cutaneous changes are accompanied by other symptoms, partly as the result of fever, partly of infection. The sensorium is often affected, and delirium or somnolence and coma may appear very early. The tongue is often dry, fissured, brownish-yellow, or covered with sordes, as in typhoid fever. Catarrhal angina is often present. The patients suffer not infrequently from repeated vomiting, and complain of gastric pain. The spleen is often, the liver less frequently, swollen and painful. The bowels are usually constipated, diarrhœa is rare. Febrile albuminuria is common. The urine presents the characteristics of febrile urine: increase of urea, and phosphoric acid. Brieger found an increased amount of phenol. Nepveu claims to have found micrococci in the blood, most abundantly in the blood of those parts which were attacked by the erysipelas.

The disease sometimes lasts only a few days, in other cases several weeks or even months.

Among the rarer complications are changes in the skin itself, such as herpes facialis, roseola, or impetigo. In some cases, there are multiple cutaneous abscesses, after opening which the erysipelatous changes may subside in a sort of crisis. In one case, Holms described vasomotor disturbances (bluish-red color and anæsthesia) of the phalanges of the fingers, followed during convalescence from the erysipelas by spontaneous gangrene of the parts in question.

Fatal purulent meningitis sometimes develops during an attack of erysipelas. Gangrenous erysipelas near the eyes may terminate in panophthalmitis and phthisis bulbi.

Accidental (febrile) cardiac murmurs are not uncommon. Verrucose or ulcerative endocarditis, myocarditis, and pericarditis sometimes develop. Jaccoud maintains that pericarditis never occurs without endocarditis, and that the latter attacks only the venous valves.

Sudden œdema of the glottis occasionally puts an end to existence. Bronchitis, pneumonia, pleurisy, and mediastinitis are occasional complications. Violent epistaxis occurs in some cases as a sort of crisis.

Icterus, dysenteriform stools, and intestinal hemorrhages are occasionally observed as the result of round duodenal ulcers or ulceration of the intestinal lymph follicles. Peritonitis has been described in a few cases, especially in erysipelas of the abdominal walls.

Acute (hemorrhagic) nephritis may develop, and sometimes terminates in chronic Bright's disease. Bahde observed glycosuria which lasted three days.

Cicatrices sometimes remain as sequelæ, particularly in gangrenous erysipelas which has led to deep destruction of the skin. When erysipelas often relapses in the same locality, it is apt to give rise to hyperplastic and hypertrophic changes in the subcutaneous connective tissue (elephantiasis Arabum). Anæsthesia or hyperæsthesia is sometimes

observed on the site of erysipelas. Obstinate neuralgias are sometimes left over. In one case, Broadbent described atrophy of the skin associated with anæsthesia. Defluvium capillitii is quite a constant symptom of erysipelas of the scalp, on account of nutritive disturbances of the hair follicles, but the hairs generally grow again after a certain lapse of time.

Joint changes have been described in a number of cases. They consist of pain, or multiple painful swellings, or of purulent inflammation. Ritzmann observed a fatal termination in two of these cases. Purulent parotitis is mentioned in a few cases.

The ocular changes are important. Abscesses sometimes form in the

Fig. 22.

Erysipelas cocci in the cutis. Enlarged 750 times. Immersion lens.

lids, or the latter are partially destroyed by gangrene of the skin. There may also be inflammation of the orbital cellular tissue which, in Knapp's case, gave rise to compression and thrombosis of the retinal vessels. Keratitis is an occasional sequel. Amaurosis sometimes develops very rapidly, and is found to be the result of atrophy of the optic nerve and retina. Opacities of the vitreous humor and glaucoma have also been described. These changes sometimes undergo gradual resolution.

Erysipelas of the mucous membranes may develop primarily and gradually spread to adjacent integument, or it is secondary to erysipelas of the skin. The disease is occasionally confined to the mucous membrane, but it may then be difficult to recognize its character. It occurs most frequently in the pharynx, next in the nasal mucous membrane or on that of the lachrymal passages. In the puerperal state, it sometimes

develops on the mucous membrane of the vagina and uterus, whence it may pass through the tubes to the peritoneum and give rise to peritonitis.

The principal changes are swelling and redness of the mucous membranes, inflammation of the adjacent lymphatic glands, formation of vesicles and abscesses. The diagnosis is rendered positive if erysipelas of the skin is also present. The clinical history is the same as that of cutaneous erysipelas.

III.—Anatomical Changes.—There is no longer a doubt that erysipelas owes its origin to the proliferation of lower organisms. The erysipelas cocci are round bacteria (micrococci) which are often arranged in pairs or in chains of six to twelve (vide Fig. 22). Fehleisen cultivated them and inoculated them successfully in human beings. The size of the cocci is 0.3 to 0.4 μ. They are found only in the lymph vessels of the skin, more rarely in the lymph spaces, but not in the blood-vessels. They appear only at the borders of the erysipelatous patch, and in the healthy periphery, not in the most intensely affected portions of the skin. Numerous round cells are found along the lymphatics, and the blood-vessels are congested. The changes affect not alone the cutis proper, but also extend irregularly into the subcutaneous adipose tissue.

It must be remembered that in the corpse the redness of the skin disappears, leaving only the swelling.

The general symptoms depend, apart from the fever, perhaps only secondarily upon certain toxic substances which are produced by the active proliferation of the micrococci, since Fehleisen never found bacteria in the blood.

The changes in the viscera include enlargement of the spleen, cloudy swelling of the heart, liver, and kidneys, swelling of the intestinal lymph follicles, and, at times, ulcerations of the intestinal mucous membrane.

Klebs and Reiner have raised the question whether erysipelas is always produced by certain definite bacteria. In the erysipelatous skin of patients suffering from typhoid fever, they found bacilli, and believe that these bacilli, if carried into the lymphatics of the skin, may set up erysipelas.

IV. Diagnosis.—The disease is easily differentiated from others by the redness, heat, and swelling of the skin, inflammation of the adjacent lymphatics and glands, and the serious impairment of the general condition.

It is distinguished from cutaneous phlegmon by the fact that in the latter the infiltration of the skin is as hard as a board, and exhibits a tendency to the formation of abscesses. In acute purulent œdema, the skin also exhibits a doughy infiltration, but general symptoms often remain absent for a long time.

V. Prognosis.—In uncomplicated erysipelas the prognosis is not very bad, and many individuals survive a large number of attacks. But it should not be forgotten that unforeseen complications may arise, and that the severity of the general symptoms, particularly in the old and decrepit and those addicted to alcohol, sometimes result with astonishing rapidity in a fatal termination. Rapidly fatal collapse is not infrequent in gangrenous erysipelas, and the disease is also very grave in puerperal women.

VI. Treatment.—As a prophylactic measure, all wounds, however slight, must be thoroughly disinfected with carbolic acid (five per cent)

and kept scrupulously clean. In hospitals, strict attention must be paid to ventilation and cleanliness as regards clothing, bedding, bandages, walls, and floors. During endemics or epidemics, all operations, even vaccination and hypodermic injections, must be avoided. The erysipelas patients should be isolated and have separate attendants.

Erysipelas requires local and general treatment. The best local application, in our opinion, is carbolic acid dissolved in turpentine (℞ Acid. carbolic., ʒ ss.; ol. terebinth., ℥ i. M. D. S. To be applied externally every hour). It should not be applied in places where vesicles have burst, and the skin is deprived of epidermis. The application should be made two to five centimetres beyond the borders of the erysipelatous region. If the skin is very tense, small incisions may be made to relieve the tension and prevent gangrene. But if gangrene occurs despite our efforts, the part should be dressed with acetate of alumina (one to two per cent).

The patients are kept in bed in a large, airy room, receive fluid food, and lemonade to relieve thirst; a daily evacuation from the bowels should be secured. Fever is best treated with antipyrin (ʒ i.–iss. by enema). In many cases, the disease runs its course spontaneously in a few days, so that antipyretic measures are not always necessary.

Complications require purely symptomatic treatment.

The following local measures have also been recommended: subcutaneous injections of carbolic acid (one to two per cent) especially in the still healthy surrounding parts; application of turpentine every ten to fifteen minutes; ferric chloride solution, collodion, tincture of iodine, faradism (!), æther douche, ice-water or lead-water compresses.

Withers recently extolled the internal use of potassium iodide (gr. vij. every two hours).

6. *Herpes.*

Herpes gives rise to the formation of small vesicles which are arranged in groups upon a reddened base. At first the contents of the vesicles are clear and serous, later they grow cloudy and pus-like, and in two to four days dry into thin crusts. The latter fall off without leaving a scar, but the spot remains red and pigmented for some time. Many vesicles are more or less umbilicated.

Herpes may appear upon the integument or mucous membranes (soft palate, prepuce, conjunctiva, tongue, larynx, cornea).

The opportunity for the development of herpes is always afforded by inflammations of peripheral nerves. These may be the result of injury, compression, etc., or of infectious agents. The latter may be primary or secondary, according as we have to deal with an independent infectious disease or with a complication with a previously existing infectious disease.

a. Herpes Facialis.

I. Symptoms and Etiology.—Zimmerlin observed an instructive series of cases of primary infectious herpes facialis in the Basle Citizen's Hospital. Thirty patients—all in the same wing of the hospital—were attacked within three months.

The secondary infectious form is more frequent. It is observed in many febrile infectious diseases, most frequently in fibrinous pneumonia, also in relapsing fever, malaria, meningitis, but only in exceptional cases

in typhoid or typhus fever. The vesicles develop generally at the boundary between the skin and the mucous membrane of the lips (herpes labialis). As a rule, only one-half the lip is affected, more rarely it extends over the entire upper or lower lip, or on one side of both lips. Occasionally the entire circumference of the mouth is surrounded by groups of vesicles; the lips are then swollen, and later become covered with more or less thick, gray, brown, or bloody crusts. In certain cases, the vesicles also appear, at the same time, on the same side of the mucous membrane of the cheek, or the hard and soft palate. Unilateral herpes of the tongue (glossitis herpetica) has also been observed.

Herpes nasalis (on the alæ nasi), herpes auricularis (on the concha), herpes infraorbitalis, palpebralis, conjunctivalis, and episcleralis are much rarer than herpes labialis. In a case of pneumonia, Thomas observed herpes sacro-ischiadicus, in another case herpes facialis and herpes manus.

Herpes facialis also occurs not infrequently in gastric catarrh, also in some women at the period of menstruation, and occasionally as the result of violent emotions.

Nothing is known with certainty concerning the relation between herpes and febrile infectious diseases. According to Gerhardt, the fever gives rise to dilatation of the blood-vessels, and thus to mechanical irritation of branches of the trigeminus within narrow and unyielding bony canals in the skull. But since all febrile diseases do not give rise to herpes facialis with like frequency, it seems more plausible to us to assume that a neuritis is associated with the infectious process, and that the bacteria of one infectious disease find a more ready entrance to the peripheral nerves than those of another disease.

II. Treatment.—Treatment is hardly necessary. If the entire circumference of the lips is affected, painful rhagades sometimes form; these may be brushed with ol. amygdalar. every two hours to accelerate the exfoliation of the crusts. If the patients complain of pain in the mouth, bad taste, and fœtor ex ore on account of vesicles in the buccal cavity, we may order a gargle of liq. alumini acet. (5 : 100, one tablespoonful in a cup of lukewarm water) every two hours. If potassium chlorate is preferred, it should not be given in too concentrated a solution (5 : 200).

b. Herpes Zoster. Zona.

I. Etiology.—It has long been known that some forms of herpes zoster are infectious in their origin, since it often occurs in epidemics during the spring months, and generally attacks an individual only once in a life-time.

In addition, there are non-infectious forms of herpes zoster. They are associated most frequently with nervous diseases (brain, spinal cord, and peripheral nerves), either from disturbances of certain trophic nerve tracts, as in diseases of the brain and spinal cord, or from direct injury to the peripheral nerves. Thus, herpes zoster often occurs in cancer or tuberculosis of the spine if the intervertebral ganglia, which possess trophic functions, are compressed and inflamed. It appears not infrequently in phthisis when it becomes complicated with tuberculosis of the vertebræ. Among 1,000 cases of phthisis, Leudet observed 17 of herpes zoster. In other cases, affections of the peripheral nerves can be demonstrated, for example, in adhesive pleurisy, mediastinal tumors, aneurism of the aorta, injury to the peripheral nerves, or compression

by callus. According to David, dental operations or injuries may cause herpes of the cheeks and gums, and this may also be produced by the eruption of a wisdom tooth. In two cases, Gerhardt observed herpes of the chin after galvanization of the mental nerve.

The disease has been observed after poisoning with carbonic oxide and after the use of arsenic. Hutchinson attributes some cases to syphilis, and maintains that this is especially true of bilateral herpes. Hauff observed herpes zoster after acute articular rheumatism. According to some writers, pregnancy predisposes to herpes.

The disease occurs at every age (Boehm observed it in two infants aged 5 and 7 months respectively), but it is most frequent from the twelfth to twenty-fifth years.

II. Symptoms.—In herpes zoster, the herpes vesicles develop along the course of certain nerves. It is generally unilateral, but occasionally even multiple, *i. e.*, it appears along several distinct nerves. It is most frequent upon the trunk.

It may be divided into different varieties : herpes zoster capillitii, faciei, nuchæ, brachialis, pectoralis, abdominalis, and femoralis.

The most frequent form is herpes zoster pectoralis. The vesicles generally occupy one to four intercostal spaces. In typical cases, they begin at the spine, then descend along the course of the intercostal nerves, and on the anterior part of the thorax again descend. They very often extend a little beyond the median line, both anteriorly and posteriorly. In some cases the groups of vesicles are scattered, not connected.

The changes begin with diffuse erythema, upon which develop small red papules, which are soon converted into clear vesicles, from the size of a pin's head to that of a lentil. The individual vesicles sometimes coalesce. The vesicles belonging to one group pass through all the changes simultaneously, but different groups often appear at different times. Fully developed vesicles may not form in certain groups. In others, the vesicular contents assume a hemorrhagic black color, and deep destruction of the cutis and cicatrices result. In such cases the disease may last two or three months, while it generally runs its course in two to four weeks.

The eruption is often preceded by prodromata. In herpes zoster pectoralis, intercostal neuralgia develops, and may last four to six weeks. Chill, fever, and gastro-enteritic symptoms may appear a few days previously. Furthermore, the patients complain not infrequently of pain, tremor, and spasm in certain muscles. In herpes zoster of the scalp or face, delirium and vomiting have been observed in addition to neuralgia of certain branches of the trigeminus.

The eruption rarely appears without prodromata. The patients complain of severe stitches and painful prickling in the skin, and then notice the eruption.

The neuralgia and fever often subside after the appearance of the vesicles. In other cases, the neuralgia increases and gives rise to obstinate insomnia and nocturnal excitement. In the most favorable cases, the vesicles heal in one to two weeks. The disease lasts longer if the different groups of vesicles follow one another at long intervals, or if hemorrhages occur into the vesicles with extensive destruction of the skin.

Herpes of the first branch of the trigeminus (herpes ophthalmicus) is

sometimes attended with severe ocular diseases. In 80 cases collected by Cocks, the eye was affected 46 times (left eye 40 times). The ocular complications consist of herpes upon the conjunctiva and cornea, anæsthesia of the cornea, iritis, diminution of intraocular pressure, even panophthalmitis. Hutchinson states that ocular complications never occur unless herpes vesicles appear upon the upper part of the nose, but several exceptions to this rule have been observed. In herpes zoster of the second branch of the trigeminus, vesicles form upon the mucous membrane of the cheeks, hard and soft palate. In some cases, the herpes of the mucous membrane is primary, and is followed by herpes of the face, or the former may exist alone. It is sometimes followed by falling out of the teeth and atrophy of the jaw.

Sequelæ are not very rare. They include obstinate neuralgias which may not begin until the vesicles have healed. Paralysis of the facial nerve or the limbs has been observed, but almost always disappears at the end of a certain time. Duncan reports hemiplegia in two old women, and explains it as the result of a reflex influence exerted through the sympathetic (?). Atrophy of the muscles, hyperhidrosis, anhidrosis, anæsthesia and paræsthesia, and falling out of the hair are observed occasionally in the affected nerve tracts. Falk recently described diabetes mellitus as a sequel of herpes zoster.

II. Anatomical Changes.—In herpes zoster pectoralis, Baerensprung first observed inflammation of the intervertebral ganglia. In a case of herpes ophthalmicus, Wyss found inflammation of the Gasserian ganglion. These lesions tally with the theory of the trophic properties of the parts in question.

As a matter of course, herpes zoster will also develop if the trophic fibres in the peripheral nerves are inflamed, and this lesion has been found in some cases. In the vicinity of the vesicles, however, secondary changes may develop in the cutaneous nerves.

The following are the stages of development in the anatomical structure of the vesicles: dilatation of the vessels of the cutis, serous exudation, emigration of white and a few red blood-globules—elevation of the epidermis from the rete Malpighii by serous exudation, and the formation of vesicles (the latter are chambered, the individual septa consisting of compressed epidermis cells)—formation of nests of wandering cells within the rete Malpighii by division of the epithelium cells, transformation and partial destruction of their protoplasm—drying of the fluid contents and regeneration of the stratum corneum through the agency of the remaining rete Malpighii.

III. Diagnosis, Prognosis, Treatment.—The diagnosis is easy if we bear in mind the characteristic grouping of the vesicles, and their distribution along definite nerve tracts.

The prognosis is almost always favorable. Death occurs only in exceptional cases, as in Wyss' cases, in which it followed panophthalmitis.

Treatment should not be active. The vesicles should be brushed every morning with ol. olivar., ol. amygdalar., or ol. hyoscyami, covered with cotton batting, and protected against pressure and friction. Subcutaneous injections of morphine may be made in violent neuralgia. If the neuralgia persists after the vesicles have healed, we may order quinine (gr. 15 to 30), Fowler's solution (5 to 10 drops t. i. d.), the constant current, or morphine subcutaneously. The faradic current may be employed if paralysis and atrophy ensue.

c. Herpes Progenitalis.

I. Symptoms and Diagnosis.—In herpes progenitalis, the vesicles appear on the genitals. As a rule, they are few in number. In men, they are most frequent on the inner surface of the prepuce, but also appear on the glans and dorsum penis. They appear rarely on the anterior part of the urethra, and may then give rise to blennorrhoic discharge. In women, they appear on the prepuce of the clitoris or inner surface of the labia minora, rarely on the labia majora.

The patients complain generally of a peculiar prickling and itching. Examination usually shows shallow ulcers but no vesicles, which may increase in size if the patients have yielded to the desire to scratch. They then form deep, suppurating losses of substance, which may readily be mistaken for soft chancre, or, if the base is inflamed, for hard chancre. Balanitis, balano-posthitis and œdema of the prepuce may also develop. The true explanation would be apparent at once if the lesions appeared spontaneously and without previous intercourse, but the statements of the patient cannot be relied upon in this regard. Enlargement of the inguinal glands would favor the diagnosis of chancre. Finally, herpes heals in a few days without special treatment.

II. Etiology.—Some individuals suffer from herpes progenitalis after almost every act of coitus. It occurs often in cases of phimosis or if the smegma is abundant. It may appear in epidemics, particularly in the spring. The disease exhibits a great tendency to relapse.

III. Treatment.—The individuals must abstain from coitus until the eruption is entirely healed. The sores are covered with borax vaseline, carbolized oil, or zinc ointment. Phimosis, or an abundant production of smegma must receive suitable treatment.

d. Herpes of the Pharynx.

(*Angina herpetica.*)

I. Etiology.—Herpes sometimes develops in the pharynx as an independent affection. It is attributed to colds and menstrual disturbances (at the onset of the menses). A few years ago, I observed an epidemic of pharyngeal herpes in Goettingen, and at the same time herpes præputialis and erythema nodosum were also remarkably frequent. Herzog states that the infectious character of the disease is not demonstrable.

II. Symptoms and Treatment.—The disease often begins with a violent chill which may be followed by high fever and malaise. At the end of the first or second day of the disease, the pharyngeal mucous membrane is found to contain yellow, slightly elevated patches with red borders; their size is but little larger than that of a pin's head. They are most frequent on the arches of the palate; in one of my cases they were situated on the posterior surface of the uvula, and were only visible with the aid of the laryngoscope. They are unilateral, and thus distinguished from pseudoherpes, which forms a diffuse, irregularly distributed vesicular eruption in the pharynx, and is not infrequent in inveterate smokers. Herpes pharyngis is sometimes associated with herpes facialis, or it may alternate with herpes præputialis.

The patients complain of pain in swallowing, burning in the throat, often of a very bad taste in the mouth. The fever subsides on the third

or fourth day, the yellow patches are exfoliated, leaving behind shallow, cicatrizing ulcers. Great prostration sometimes persists for a very long time. In one case I noticed paralysis of the velum palati. Perforation of the velum palati or pillars of the palate, and pseudo-membranous inflammation have also been observed in rare cases.

Treatment: gargles of potassium chlorate (5 : 200).

e. Herpes of the Larynx. Herpes Laryngis.

(*Laryngitis Phlyctænulosa.*)

This disease is rare. It generally begins with fever, and is associated with herpes of the external skin or other mucous membranes. The laryngeal mucous membrane first presents epithelial opacities, later vesicular elevations, which terminate in shallow ulcers. The latter are surrounded by a red zone, and heal in one to two weeks. The diagnosis can only be made with the aid of the laryngoscope. Pseudoherpes may also occur upon the larynx, but, unlike herpes, it is not unilateral, and is confined to parts in which the mucous glands are abundant.

7. Febris Miliaris.

Sweating Sickness.

I. Etiology.—The existence of febris miliaris has been disputed up to the present time. From the reports of others, it appears to us that it is an independent infectious disease.

The first epidemics appeared in the fifteenth and sixteenth centuries, and then not until the eighteenth century. Since then, the disease has appeared from time to time in England, France, Italy, and Germany.

The majority of epidemics occur in the summer. Their development is favored by damp, changeable weather, perhaps by a marshy soil. As a rule, few epidemics last more than two to six weeks. It occurs most frequently between the ages of twenty to forty years.

Women are affected more frequently than men; also individuals of a strong constitution. The character of the virus is unknown, but some regard it as miasmatic in origin. The circumscribed character of the epidemics is a notable feature. The same individual may experience a number of attacks.

II. Symptoms.—The disease is preceded by general malaise for two or three days.

Patients who went to bed without special local symptoms awake during the night bathed in perspiration, together with a feeling of oppression in the præcordial region, headache, dizziness, palpitation of the heart, occasionally cramps in the calves. The temperature is more or less elevated, the pulse accelerated, respirations rapid and dyspnœal. The sweating is sometimes so profuse as to drench the bed. On the third or fourth day, miliaria appear, at first miliaria crystallina, then rubra, finally miliaria alba. The appearance of the eruption is often associated with prickling and numbness of the skin. The patients suffer from anorexia, increased thirst, nausea, vomiting, constipation, and scanty diuresis. The spleen is generally enlarged.

Death may occur in collapse or from paralysis of the heart or brain, or from complications (diphtheria, pneumonia, diarrhœa, dysentery, purpura, and dissolution of the blood).

When uncomplicated, the disease lasts six to eight days, but the period of convalescence is sometimes protracted. The disappearance of the eruption is followed by desquamation of the skin. The average mortality is ten per cent, in some epidemics as high as fifty per cent.

III. Anatomical Changes.—There is almost constantly a tendency to rapid decomposition of the body in the fatal cases, the blood is thin and black, the spleen large and soft.

IV. Treatment.—The bed covering should be light, the room kept at a temperature of 15° R., and cold water mixed with claret or brandy given as a drink; only fluid food should be allowed. To check the diaphoresis, we may give sulphate of atropia (gr. $\frac{1}{6}$: ʒ iij., one syringeful subcutaneously).

8. *Small-Pox. Variola.*

I. Etiology.—Small-pox patients are the most frequent sources of infection. In former times, before bovine virus was used in vaccination, individuals were often inoculated with the contents of pocks taken from patients, because experience taught that the disease, when acquired in this way, was more apt to run a mild course than when acquired accidentally. The scabs left over after the pustules have dried possess infectious properties, even if kept for a long time. The physiological secretions are non-infectious; whether the blood of the patients possesses infectious properties still remains doubtful.

The disease also appears to be infectious even during the period of incubation. Schaper reports a case in which a piece of skin was removed from an apparently healthy man for the purpose of transplantation. A few hours later the prodromata of small-pox appeared. After a certain period, the individual upon whom the skin had been transplanted was also attacked with small-pox.

The virus diffuses itself in the vicinity of the patient, and infects the air, articles of clothing, and utensils. The danger of infection increases the greater the number of patients in one room, and the less the room is aired. During the last great epidemics (1870 to 1873) it was noticed in some instances that when small-pox patients were placed in quarantined houses, cases developed in neighboring, but not immediately adjacent houses, particularly if the windows of the sick-rooms were constantly kept open.

Infection may also be conveyed by intermediate persons who escape the disease. The virus is generally absorbed through the respiratory organs, in exceptional cases by direct inoculation of wounds. Infection has also been observed from handling money, rags, etc., with which patients had come in contact. In many cases, it is extremely difficult to trace the mode of infection.

The virus is supposed to consist of bacteria. Cohn describes, in the contents of the pustules, fine granules which are divided in twos, fours, eights, etc., are accumulated in groups of sixteen, thirty-two, or more, and form larger zoogloœa masses. They are generally immovable, and sometimes form rosary-like chains. Klebs states that the tracheal mucus contains micrococci 0.5 μ in diameter, which are partly arranged in fours in the shape of a square, but are nevertheless separated from one another (micrococcus quadrigeminus).

The contents of the pustules may not alone infect human beings, but also certain animals (cow, calf, horse, ass, sheep, pig, dog, monkey). The body of the animal possesses the power of weakening the injurious properties of human small-pox (vide remarks on vaccination).

Almost every individual possesses a susceptibility to small-pox, but it is generally abolished or diminished in infancy by vaccination. In many persons the efficacy of vaccination does not last longer than ten years, so that revaccination should be performed at least every ten years. Very few people present permanent immunity from small-pox without previous vaccination.

In others, there is temporary immunity; they escape during one epidemic, but are attacked in the next.

As a rule, one attack confers life-long, acquired immunity, although there are occasional exceptions to the rule. It is said that some individuals have suffered from as many as six attacks.

Certain conditions may increase the susceptibility to the disease. This is particularly true of pregnancy and the puerperal state, under which circumstances the disease is often of the hemorrhagic variety.

It is claimed that drinkers, feeble individuals, and those who are exposed to heat (cooks, firemen) also exhibit a tendency to the hemorrhagic form of small-pox.

Age and sex exert no influence on the frequency of the disease. Pregnant women, suffering from small-pox, have been known to give birth to children whose skin was covered with small-pox pustules, and, on the other hand, the disease has been observed in very old people. Nor does climate exert any influence in this direction. It is said that negroes are especially predisposed to infection.

Where vaccination is common or obligatory, the disease generally occurs sporadically or is imported from without. Under other conditions it occurs in epidemics. It is said that epidemics in large cities are apt to recur at definite intervals, which vary from five to twelve years. The seasons exercise no special influence upon the occurrence of epidemics, although they are somewhat more frequent in the cold months than in the hot summer months. The spread of epidemics is sometimes favored by external conditions, for example, the crowding together of many individuals in wars under bad hygienic surroundings.

Small-pox is sometimes coincident in one individual with other infectious diseases, such as measles, scarlatina, syphilis, erysipelas, pemphigus, typhoid fever, intermittent fever, phthisis. In other cases, these diseases immediately precede or follow small-pox.

II. Symptoms.—The duration of the period of incubation varies from ten to fourteen days. After inoculation, the period of incubation is generally shorter than in natural small-pox.

Curschmann states that, in one case, the period of incubation only lasted five days, and according to Zuelzer, its average duration in malignant hemorrhagic small-pox is six to eight days.

The majority of patients feel perfectly well during the period of incubation. In a small number of cases, slight general disturbances appear towards the end of this stage: slight rise of temperature, chilliness, anorexia, headache, malaise, etc.

The prodromal stage begins, in many cases, with a single severe chill or repeated chilly sensations. The bodily temperature rises very rapidly and, in a few hours, reaches 39°, 40°, or more. At the same time, the pulse becomes very rapid (100, or even 120 beats a minute). The respirations are increased in frequency, and many patients complain of dyspnœa, generally of nervous origin. Mild delirium, epileptiform convulsions, and meningitic symptoms are apt to set in in children and excitable, feeble adults. The majority of patients complain of a feeling of dulness in the head, of vertigo of such intensity that they are unable to stand erect, and of annoying headache which, as a rule, is referred to the forehead, in some cases to the occiput, and is distributed unilaterally or along the course of certain branches of the trigeminus. The conjunctiva is often injected, and there is photophobia with increased secretion of tears. The tongue is coated, fœtor ex ore is often noticeable. Thirst is increased, the appetite is entirely lost. The patients often complain of nausea and eructations, and repeated copious vomiting occurs with unusual frequency. The bowels are constipated, the urine

scanty and saturated. The right ventricle becomes dilated; the first sound of the heart is often muffled, blowing, and replaced by a systolic murmur. The thorax may present evidences of dry bronchitis. The spleen is usually enlarged. The liver is often tender on pressure and slightly enlarged at a very early period. A very constant symptom is the violent pains in the back, which are referred to the loins and region of the kidneys. Some attribute these pains to renal congestion, others to congestion of the lumbar meninges. The latter view is more probable, because the pains often radiate into the lower limbs or abdominal walls, and are associated occasionally with anæsthesia, hyperæsthesia, or paræsthesia of the lower limbs.

Symptoms of pharyngeal and buccal catarrh (diffuse redness or hyperæmia of the mucous membrane in patches) are often found very early. Some patients suffer early from hoarseness or a burning sensation in the nose; repeated epistaxis may also occur.

In many cases, a prodromal eruption appears upon the second and third day. This may consist of diffuse erythema, as in scarlatina, of small circumscribed congestions (roseola variolosa), or of wheal-like patches. Simon showed that they are very often found on certain definite parts of the body (lower part of the abdomen, the sides of the abdomen and chest as high up as the axillæ, and the outer surface of the leg, along the extensor hallucis). Their frequency varies according to the character of the epidemic. It is not true that the site of these prodromal eruptions remains free from the small-pox eruption proper. On account of the distribution of the prodromal eruptions along certain nerve tracts, they have been attributed to paralysis of the vaso-motor cutaneous nerves.

The prodromal stage, on the average, lasts three days.

It is followed by the stage of eruption. The first cutaneous changes appear on the face and scalp, but upon the latter they are only visible if the hair is short and thin. At the end of twenty-four hours, they extend to the trunk, and finally to the limbs. The eruption also appears in the pharynx, larynx, nose, and conjunctiva, opening of the urethra, vagina, cervical portion of the uterus. The eruption passes through various stages, which will be described later.

It is a noteworthy fact that the severe general symptoms diminish very markedly with the appearance of the eruption, and many patients feel so relieved that they consider themselves recovered. The temperature falls (vide Fig. 23) and approaches the normal, and the frequency of the pulse and respirations also diminishes.

In the first or papular stage of the eruption, the face contains patches like those of measles, particularly upon the forehead and the boundary between the nose and cheeks. These patches grow pale on pressure, and are hot to the feel. In addition, the integument is œdematous, so that the eyelids are swollen and the palpebral fissure narrowed. Small patches sprout up very quickly within the patches, and are very often, though not constantly, grouped around the hair follicles or sweat glands.

Upon the trunk the patches and papules develop later, and are more scanty; they are more abundant on the limbs, particularly the extensor surfaces and on the fingers. These changes are often attended with burning and itching.

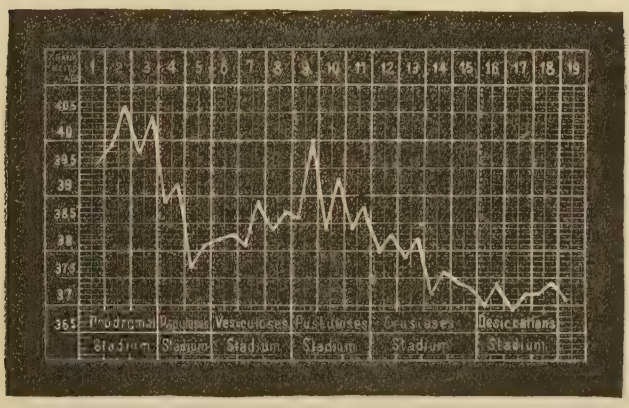
The papular stage has an average duration of two days. On the third day of the stage of eruption, *i. e.*, the sixth day of the disease, the vesicular stage begins. At the apex of the papules the epidermis is

raised from the rete Malpighii in the form of a small, transparent vesicle, at first upon the face, later upon the trunk and limbs. In the next few days, the vesicles increase gradually in size, and many of them become umbilicated.

If a vesicle is punctured, its contents escape gradually. The vesicles are often distributed along the folds of the skin or the course of the cutaneous nerves. This stage generally lasts three days.

On the ninth day begins the pustular stage. The temperature and the frequency of the pulse and respiration again increase, and chills and delirium appear in some cases. At the same time the contents of the vesicles become more and more opaque, cloudy, and purulent. Their size increases. The edge of the pustule is reddened and infiltrated, and around this there generally forms a slightly swollen, reddened zone, produced mainly by hyperæmia of the skin. As pustulation increases, the

FIG. 23.



Temperature curve of a moderately severe case of small-pox.

umbilication disappears. Pustulation begins in those parts in which the papules first appear, *i. e.*, the face. The skin is often considerably swollen, so that the patients are unable, for days, to open the eyes. The pustules are very numerous upon the fingers, and many patients complain of violent burning pains in these parts. On account of the thick epidermis, fully developed pustules are hardly ever found on the palms of the hands or soles of the feet, and the eruption appears there as red or brownish-red translucent papules, or large vesicular elevations.

In about three days, this stage reaches its end, and the stage of desiccation begins, on the average, on the twelfth day. The febrile symptoms diminish ; some of the pustules rupture, and their contents are discharged and dry into yellow, gray, and brown crusts. Some pustules do not rupture, but their contents gradually dry, the pustule becomes flabby, and finally is also converted into a crust. The majority of the patients experience intolerable itching, scratch the skin, and thus delay recovery.

The crusts gradually fall off at about the sixteenth day of the dis-

ease (stage of decrustation). Brownish-red pigmented patches remain, or, in those places in which the pustules penetrated deep into the cutis, brownish-red pigmented cicatrices form, and are converted later into white cicatrices.

The cutaneous lesions just described are associated with corresponding ones on the mucous membranes.

The pharynx is affected very early, often in the prodromal period. The mucous membrane is very red, and pustular formations develop upon it. These begin as congested papules; then the overlying epithelium is raised in the shape of a white or mother-of-pearl gray vesicle; finally, the epithelium is shed, leaving a subepithelial loss of substance. At the same time the patients complain of difficulty in deglutition, and some are hardly able to swallow even fluids.

Annoying salivation generally occurs when the buccal mucous membrane is affected. If the tongue is involved, it may increase considerably in size, and appears wedged in between the teeth (glossitis variolosa). The eruption may also form on the mucous membrane of the œsophagus.

In some cases, the changes extend to the Eustachian tube and middle ear (giving rise to tinnitus aurium, pain in the ears, etc.), and also to the mucous membrane of the air passages.

The eruption has also been observed upon the urethral meatus, vaginal portion of the uterus, and rectum.

The individual stages of the eruption are not always distinctly separated from one another, and the transition between them takes place gradually.

Variola without eruption is a very mild form of the disease. The patients, who have been exposed to infection, experience the characteristic prodromata, but the eruption does not appear, and the disease terminates with the prodromal stage.

In afebrile variola, the eruption is distinct, but there is very little or no fever. In abortive variola, the eruption terminates in the papular or squamous stage. In variola siliquosa, the vesicles contain air, but no fluid.

The majority of cases of small-pox run a much milder course than we would be inclined to believe from the above description. The milder form is called varioloid, in contra-distinction to true variola. The more vaccination is adopted the more frequently small-pox occurs as varioloid, and the more rarely as variola. As a rule, even those who have been vaccinated a long time previously suffer merely from varioloid. The latter runs a milder and more rapid course; the eruption is scanty; complications, especially severe eye diseases, are rarer; and, as a rule, unsightly cicatrices are not left over.

Hemorrhagic small-pox is the result of unusually severe, generally fatal infection. The occurrence of a few small hemorrhages between the papules is very frequent and innocuous, and does not constitute hemorrhagic small-pox. In the latter, abundant hemorrhages occur during the eruptive stage, sometimes in the shape of papules, sometimes as extensive suffusions and infiltrations, and are attended with grave collapse, hemorrhages from the nose, mouth, air passages, stomach, intestines, kidneys, and genitals. These often prove fatal before the eruption has developed. In other cases, the hemorrhagic character is manifested at a later stage. After the vesicles or pustules have formed, their contents become blackish-red and bloody, and hemorrhages also occur upon the skin and mucous membranes.

Renauld states that he has found decoloration of the red blood-globules and hæmatin crystals in the blood serum in hemorrhagic small-pox.

Confluent small-pox is another severe form of the disease. It generally begins with severe prodromata. The patches and papules are so abundant that they coalesce in places. This is true to a still greater extent of the pustules and crusts. There is very extensive inflammatory œdema of the skin. During the pustular stage, the face, forearms, and fingers are covered as if with a mask. The crusts often retain masses of pus which exude after rupture or perforation of the crust. The patients often emit an unpleasant odor. The general condition is grave: delirium, hyperpyrexia, perhaps death from heart failure. Dangerous complications often make their appearance.

The complications of small-pox attack the most various organs.

Erysipelas of the skin sometimes develops during the stage of pustulation or desiccation. Gangrene of the skin develops occasionally in the vicinity of the efflorescences. Multiple abscesses appear at times in the skin, and may be the result of pyæmic infection; this is also true of abscesses of the intermuscular connective tissue. Inflammatory swelling, suppuration, and ankylosis of the joints have been observed, particularly in the large joints of the limbs.

Delirium is one of the most frequent symptoms on the part of the nervous system. Marked psychopathic conditions occasionally develop, usually of a maniacal, more rarely of a melancholic character, sometimes associated with a suicidal tendency. These symptoms may appear before the outbreak of the eruption, or they form sequelæ and become permanent. A combination with purulent meningitis, encephalitis, encephalo-malacia, and cerebral hemorrhage is rare. A number of cases of aphasia or dysarthria following hypoglossal paralysis have been reported. Catalepsy, disseminated myelitis, acute ascending paralysis, and acute ataxia have also been observed. Peripheral paralyses are rare. Gubler and Laborde mention paralysis of the detrusor vesicæ at the beginning and also at the end of the disease. Diabetes insipidus and diabetes mellitus have also been included among the sequelæ.

Very serious changes may develop in the eye. Conjunctivitis is an almost constant symptom; this sometimes results in the formation of pus, particularly if the lids are swollen and occluded, and the removal of inflammatory products thus impeded. Hirschberg described diphtheritic patches upon the conjunctiva, which sometimes result in the loss of the eye. Small-pox pustules appear occasionally upon the conjunctiva, generally on the bulbar portion, and sometimes extend to the cornea, though they do not develop upon the latter. Adler observed pustules on the conjunctiva prior to their eruption on the skin. They are small, and form yellowish papules which are surrounded by a zone of injected vessels. Subconjunctival hemorrhages may occur in hemorrhagic small-pox, also hemorrhages into the choroid and retina. Ulcerative changes of the cornea are not uncommon. They appear most frequently as superficial circumscribed keratitis, which may terminate in hypopion, destruction of the cornea, prolapse of the iris, and thus in anterior synechia, and even phthisis bulbi. This does not often develop before the stage of desiccation. Rarer complications are diffuse interstitial keratitis and keratomalacia, the latter possessing an unfavorable prognostic significance. Iritis and irido-choroiditis are rare, and should be included among the sequelæ rather than among the complications.

Opacities of the vitreous or the posterior surface of the lens often develop. Acute glaucoma, retinitis, and neuro-retinitis have also been observed.

Aural changes are very common in small-pox. In one hundred and sixty-eight autopsies, Wendt found only two cases in which the auditory apparatus was intact. The pustules form upon the concha and the cartilaginous portion of the external auditory canal. They have not been observed upon the bony parts, the membrana tympani, internal ear, and Eustachian tube, but the mucous membrane is often congested and swollen. The hyperæmia often results in hemorrhages, and the circumscribed swelling forms polypoid proliferations of the mucous membrane. The petrous portion of the temporal bone sometimes undergoes inflammatory changes.

In addition to inflammation, pustulation, and hemorrhage into the nasal mucous membrane, ulcerations of the mucous membrane, bones, and cartilages sometimes develop and give rise to adhesions, deformity, and disturbances of function.

Inflammation of the salivary glands is not very common. The salivation which is observed so often is usually a reflex symptom of inflammation of the buccal mucous membrane. Metastatic inflammations of these glands may occur in pyæmic conditions (generally, however, as sequelæ), and may be associated with phlegmonous changes in the submaxillary cellular tissue. Noma, retropharyngeal abscess (sometimes followed by erosion of the carotid and death from hemorrhage), necrotic or diphtheritic changes in the pharynx, and phlegmonous œsophagitis have also been observed.

Bronchitis is such a constant symptom that many writers do not look upon it as a complication. Laryngeal catarrh is also very common. The situation becomes very grave when signs of œdema glottidis appear or necrotic changes develop in the laryngeal cartilages. The latter may prove fatal at a late period, or produce permanent hoarseness as the result of ankylosis and deformity. When the bronchitis extends to the finer air passages, it is sometimes complicated with broncho-pneumonia; fibrinous and hypostatic pneumonia or pulmonary gangrene occasionally develop. Œdema of the lungs is sometimes the immediate cause of death. Pleurisy, very often purulent, is not a rare complication. Dangerous pulmonary hemorrhages occur in hemorrhagic small-pox.

Pericarditis may develop independently, or it is secondary to pleurisy. Endocarditis, occasionally septic in character, is sometimes observed. The frequency of myocarditis has been greatly exaggerated in some cases. According to Verstraeten, the white blood-globules increase, the red globules diminish so much more rapidly in numbers the more violent the disease.

Hæmatemesis is not infrequent in hemorrhagic small-pox. Diarrhœa is a not favorable complication, and the bloody stools of hemorrhagic small-pox have a bad prognostic significance.

Menstrual disturbances are frequent in women. The menses generally appear too early or too abundantly. Pregnancy is a grave complication, since the patients are attacked, with relative frequency, with the severest forms of small-pox. Premature delivery or abortion is frequent. In rare cases, the children present the small-pox eruption at birth; more frequently they are attacked a few days later.

It is sometimes found that, in twin births, only one child is attacked. Cases

have also been reported in which the mother was healthy, but the child was attacked with small-pox. It is probable that in such cases the disease was overlooked in the mother.

Orchitis has been observed in men; it is either parenchymatous or affects chiefly the coverings of the testicle.

Albuminuria is very common, and appears at times in the initial period; it may be febrile in character or the result of infection. Acute nephritis is not very common. Hæmaturia has been observed in hemorrhagic small-pox.

During the febrile period, the urine presents the ordinary characteristics of febrile urine. In one case of hemorrhagic small-pox, Brieger found a very small amount of phenol in the urine. Leucin, tyrosin, and fatty acids have been found in the urine.

The complications and sequelæ of small-pox cannot be sharply distinguished from one another. Cutaneous cicatrices not infrequently persist for life, and sometimes cicatricial tumors (keloid) form, and must be removed with the knife. The hair often falls out, as after other severe diseases, but if the pustules have destroyed the hair follicles, restoration of the hair does not take place. The nails may be exfoliated.

Pemphigus and acne rosacea have been described as sequelæ. Cachectic œdema may develop, occasionally œdema of one limb from marantic thrombosis of the limbs. Paralyses, disorders of the special senses, diseases of the heart and kidneys, may also be left over. Psychopathy may also develop. In one of my cases violent maniacal attacks occurred from the start, and persisted after recovery from the small-pox. Phthisical processes in the lungs develop occasionally as sequelæ. Small-pox cicatrices in the œsophagus may produce constriction of that organ.

The disease very rarely attacks the same individual twice, and still more rarely do relapses occur. In Michel's two cases, another violent outbreak of the eruption appeared upon the eighteenth and twenty-second days.

Small-pox sometimes exercises a favorable influence on other diseases, for example, chorea and whooping cough.

III. Anatomical Changes.—Bacteria play a part in the anatomical changes of small-pox. They are found in the superficial and deep layers of the corium, either arranged in groups or in a tube-like shape, and occasionally in the blood-vessels. In the later stages of the disease, they disappear. The blood-vessels of the cutis dilate in places (patches of hyperæmia). Coagulation necrosis of the epithelium cells occurs beneath the groups of bacteria in the deepest layers of the rete Malpighii, and the cells are converted into non-nucleated clumps. At the same time, there is a circumscribed increase in size of the epidermis (papules). Between the necrotic cells are formed cavities, filled at first with serous, later with more purulent fluid. The necrotic cells form a mesh-work in which the fluid is inclosed. The meshes are often least distensible in the centre, so that the surface of the epidermis becomes depressed and umbilicated. The umbilication may also be produced by the development of the efflorescence around a sweat gland or hair follicle, the epidermis remaining firmly adherent in such places, and not easily removed as at the periphery. According to Auspitz and Basch, swelling of the periphery may also produce apparent depression of the

centre of the efflorescence. According to Unna, the pock first forms between the layers of the stratum lucidum.

It is only when the efflorescence extends into the cutis that permanent deep cicatrices are left over.

Bacteria have also been found in the lymphatic glands, liver, kidneys, and spleen. They are often surrounded by a double-contoured membrane, and in the liver and kidneys were situated in veins and capillaries (loops of the glomeruli). The surrounding cells are either intact or in a condition of coagulation necrosis; there is sometimes an accumulation of round cells in the vicinity.

The muscles often have the color of ham, and may be in a condition of cloudy swelling and wax-like degeneration.

The blood is sometimes fluid and has a tarry color.

The spleen is enlarged and soft; on section its follicles are often found to be unusually large.

The heart, liver, and kidneys are generally in a condition of cloudy swelling or fatty degeneration. Necrotic and diphtheritic changes are found in the intestinal mucous membrane. The mesenteric glands are often swollen.

Oöphoritis and perioöphoritis are observed not infrequently.

Small-pox efflorescences have been found on the mucous membrane of the larynx, trachea, bronchi, œsophagus, and stomach.

In hemorrhagic small-pox, extravasations of blood are found in various organs, even in the medulla of the bones and sheaths of the peripheral nerves. The cutaneous hemorrhages are the result of diapedesis of red blood-globules.

The corpse remains infectious, probably on account of mechanical desquamation of the skin.

IV. Diagnosis.—The diagnosis is easy, particularly during an epidemic. Under other circumstances, it may be mistaken for impetigo contagiosa, but in the latter the general condition is less affected, pain in the back and vomiting are absent in the initial stage, and recovery is more rapid. In the variola-like eruption produced by inunction with tartar emetic ointment, the general condition is not seriously affected. Similar eruptions caused by syphilis disappear rapidly under the use of potassium iodide and mercury.

If epidemics of small-pox and typhoid fever are prevalent at the same time, violent pains in the back during the prodromal stage favor the diagnosis of small-pox, pains in the legs that of typhoid fever.

The differentiation between measles and small-pox is difficult or even impossible during the papular stage. We must then wait twenty-four hours, and if vesicles then appear upon the papules and if the mucous membrane of the mouth and pharynx contains vesicles, a positive diagnosis of small-pox may be made.

V. Prognosis.—The prognosis is best in varioloid, more serious in confluent small-pox, most grave in the hemorrhagic form. Old and decrepit individuals and drunkards possess slight resisting powers. Pregnancy is a grave, often fatal complication.

VI. Treatment.—Prophylaxis includes not alone the strict isolation of the patient, but also the abolition or diminution of the susceptibility to the disease by vaccination with bovine lymph. Revaccination should be performed at least every ten years, if possible every five years.

Isolation is best secured in small-pox hospitals. The patient's house and furniture should be disinfected with sulphurous acid, the clothing

and bedding by dry heat or carbolic acid (five per cent). If the patient is kept at home, all those dwelling in the house should be quarantined, and later the entire house must be disinfected. The patient himself may not communicate freely with the outside world until desquamation has entirely ceased, and before several warm baths have been taken.

Small-pox corpses should be placed in hermetically sealed coffins, and then buried as quickly as possible in vaults. The funeral should be strictly private.

The treatment of the disease itself is purely dietetic and symptomatic.

The patient should have an airy room, at a constant temperature of 15° R. The diet should be fluid: milk, eggs, soup, wine; a daily evacuation from the bowels should be secured.

A protracted lukewarm bath (28° R.) should be taken morning and evening.

Disturbances of deglutition may be treated with gargles of potassium chlorate (10 : 200) after each meal; acetate of alumina (one per cent) is preferable if there is fœtor ex ore. Pieces of ice may be swallowed by the patient.

If there is constant high fever, lukewarm baths (26° R., twenty to thirty minutes) are indicated, in combination with antipyrin (℥ i.–iss. by enema).

If there is violent delirium, headache, and meningitic symptoms, an ice-bag to the head is indicated.

Hemorrhagic small-pox requires stimulating treatment: large doses of alcohol, ether, camphor, or musk, with hæmostatics.

The following measures have been recommended to favor the healing of the pustules and prevent the formation of cicatrices: application to the face of indifferent fats, collodion, tincture of iodine, carbolic acid solution, incision of the pustules and cauterization with nitrate of silver. Cold compresses or warm poultices are the most agreeable to the patient.

9. *Vaccination.*

I. Variolation consisted in the inoculation of healthy individuals with the contents of small-pox pustules. This was done either through means of a puncture or cut beneath the epidermis, or the contents of the pustule were applied to the corium, which had been laid bare by means of a fly blister.

But the dangers of this procedure must not be underestimated. According to Ferro, there was one fatal case among eighteen variolations, according to Wilson, one in 662 cases.

A still graver objection was the danger to surrounding individuals. A few cases of variolation afforded a favorable opportunity for the outbreak of an epidemic of small-pox. Hence it was entirely justifiable to prohibit variolation by statute.

II. Exanthemata, like small-pox, also occur in certain domestic animals, such as sheep, horses, and cows (ovinola, equinola, vaccinola).

Sheep-pox (ovinola) is very similar in its clinical history to the small-pox of the human species. It begins with general febrile disturbances, leads to an eruption of pocks on the skin, is conveyed through the air to other animals, and often produces great mortality. To diminish its dangers, farmers inoculate healthy animals with the contents of the

pocks, but this is only permitted at the approach of an epidemic, because the inoculated flocks may prove a source of infection for healthy ones.

In horse-pox (equinola), a pustular eruption appears upon the pasterns, and general symptoms are wanting or very slight.

The mildest course is run by cow-pox (vaccinola). This is localized on the udder, especially on the dug. The infectious matter is present in the contents of the pustules alone, and is not conveyed through the air. General disturbances are entirely absent.

The contents of animal pocks may be inoculated successfully in man, monkeys, the camel, ass, pig, goat, dog, cat, and rabbit.

The contents of human pocks, when inoculated into animals, always produce that form of the disease which is peculiar to the animal in question. If a cow is inoculated with variola-lymph, a pustular exanthem is alone produced on the udder of the animal, and if the contents of the latter are reconveyed to man, the same effects are produced as by vaccination with the natural cow-pox.

This has been denied by Chauveau and other physicians of Lyons. They inoculated cows with variola, and, on revaccination, produced variola in children.

Some authors maintain that all forms of animal pox and human small-pox are the products of the same virus. This has been supposed to consist of certain micrococci, although inoculations with artificial cultures have not proven successful. The close relationship between animal and human pox is also shown by the fact that vaccination of human beings with any form of animal pox prevents infection with human pox.

III. The inoculability of man with cow-pox has long been known. Farmers were long aware that milkmaids were apt to suffer from pustular eruptions on the fingers if they milked, with sore fingers, cows whose dugs were covered with pox pustules. It was also known that, during epidemics of small-pox, those individuals who had been infected with cow-pox either escaped or suffered very slightly. Jenner first proved scientifically that vaccination guards against variola. On May 14th, 1796, he inoculated James Phips with the contents of cow-pocks taken from the arm of the milkmaid Sarah Nelmess. After the cow-pox pustules had healed, he inoculated the boy with the virus of small-pox and found that the latter was inactive.

IV. Three varieties of bovine lymph may be employed in vaccination, viz.: humanized lymph, primary bovine lymph, and retrovaccination lymph.

Humanized bovine lymph is the term applied to the contents of pustules of cow-pox, produced by previous vaccination in man. Vaccination may be performed from arm to arm, or with artificially preserved, humanized bovine lymph. Formerly, the contents of the pustules were dried between cover glasses, or coated over whalebone tips, but this has given way, in great part, to preservation in fine glass tubes, which possess a spindle-shaped dilatation in the middle. The lymph is collected in a carefully cleaned watch glass, the two closed ends of the tube are then broken off with the fingers, and the tube held almost horizontally, with one open end in the fluid. The tube then fills with lymph, and the ends are again closed over a spirit lamp. Care must be taken that the tube contains no air, in which event fungi are apt to develop and decompose the lymph. The efficiency of the lymph may not be relied

upon for more than six to twelve months, and it should be kept in a cool, dark place. The lymph is ineffective if it contains opacities and clouds.

It should be taken only from healthy children who come of healthy families. Special attention must be directed to the presence of phthisis, scrofula, or syphilis in the family. The pustules, whose contents are gathered, must possess all the characteristics of a regularly developed cow-pox pustule. Those which possess a very broad zone of inflammation must be avoided, since vaccination with their contents sometimes causes extensive inflammation, and even erysipelas. The pustules should be taken from children who are vaccinated for the first time, since the lymph from pustules of revaccinated persons is less certain in its effects, and often produces doubtful efflorescences. The best time for collecting the contents of the pustules is the eighth or ninth day after vaccination. Under no circumstances may lymph be employed which contains macroscopic amounts of blood. If the child unfortunately happens to be syphilitic, the simple contents of the pustule will not convey syphilis, while this may be expected if there is a distinct admixture of blood with the lymph. On microscopical examination, however, all lymph contains a few red-blood globules.

Mueller's glycerin bovine lymph has been much discussed. Mueller found that if the lymph is greatly diluted with glycerin it loses none of its virtues, and these become more permanent. He recommended that the pustule be opened, its contents removed with a clean brush, placed in a carefully-cleaned watch-glass, and mixed with four times the amount of a mixture of equal parts of glycerin and distilled water. This is kept in glass tubes. An addition of sulphate of soda, salicylic acid, or thymol has been recently recommened to increase the stability of the properties of the lymph.

It has been claimed that the employment of humanized bovine lymph may convey diseases to healthy children, and also that, if the vaccination is continued from man to man, its efficacy would finally be exhausted. The first objection is met by employing great care in the choice of the child from whom the lymph is derived. It is true that some children suffering from hereditary syphilis present no signs until the age of three months, but danger in this regard is avoided by employing children who are past the age of six months. Experience in no wise justifies the fear of the gradual impairment of the efficiency of the lymph.

The employment of primary bovine virus is also not free from objections. In the first place, the disease itself is not very common, so that the lymph cannot be obtained whenever desired, and in addition, the primary lymph may produce violent symptoms in man; often great swelling of the arm, erysipelatous redness, and not inconsiderable febrile movement. If the animals suffer from garget, the inoculation of tuberculosis is possible, so that the animal should be killed, and the healthy condition of its organs rendered certain before vaccination is performed. The symptoms are milder if the animal has been inoculated by vaccination with primary bovine lymph.

In retrovaccination, the bovine lymph is revaccinated from man upon the udder of calves, and vaccination in man is then continued with the contents of the pustules produced in the animals by the vaccination. In this case, also, only those animals should be employed who present healthy viscera after death.

The virus of bovine lymph probably consists of bacteria, but these are not known with certainty. Osrich recently claims, however, to have successfully employed artificial cultures in vaccination.

V. In vaccination by puncture, a lancet is inserted quite horizontally beneath the epidermis, so that it enters between the horny and mucous layers as near as possible to the corium. Superficial injury of the latter, and the appearance of a small drop of blood are immaterial, but deep perforation of the cutis is to be avoided because this is apt to be followed by the formation of a furuncle. The tip of the lancet is then smeared with lymph, and the latter wiped off into the opening of the puncture.

Others prefer incisions which should be made close to the cutis: crossed incisions may be recommended. Some scrape off the epidermis superficially, and then introduce the lymph. Special apparatus for vaccination is, to say the least, unnecessary.

Many physicians claim that a single well-developed pustule is a sufficient guard against small-pox. Our personal preference is for several vaccinations, but we believe that three or four will suffice.

Girls are often vaccinated upon the thighs in order that the cicatrix may not be visible. We generally vaccinate on the outer side of the arm, a little below the insertion of the deltoid. The sites of vaccination should be about two centimetres distant from one another to prevent coalescence of adjacent pustules.

As a general thing, children should be vaccinated after the age of six months, although vaccination may be performed without risk even in the new-born. If there is danger of the outbreak of an epidemic, the children should be vaccinated irrespective of age. Those who suffer from rickets or scrofula, or who are teething, and feeble, anæmic children should not be vaccinated until health is restored.

The instruments and lymph should be kept scrupulously clean: if a large number of vaccinations are performed at one time, the lancet should be cleaned in a five-per-sent solution of carbolic acid after each vaccination, and the arm of the vaccinated individual should also be cleaned with a solution of carbolic acid before the operation.

Intrauterine vaccination is the term applied to vaccination of pregnant women, after which it is claimed that vaccination of the child does not take. According to the majority of recent writers, this method is, to say the least, unreliable.

VI. The immunity conferred by vaccination diminishes year by year, and cannot be assumed to last longer than ten years. Hence the necessity of re-vaccination.

During epidemics of small-pox, every one should be vaccinated if a longer interval than ten years has elapsed since the last vaccination, or if previous vaccinations have not taken. In many cases, vaccination takes even after much shorter intervals. On the other hand, vaccination, in rare cases, never takes. Immunity against vaccination is sometimes presented by children whose mothers suffered from small-pox during pregnancy. In such cases, the new-born may or may not present cutaneous evidences that they have suffered from variola in utero.

Vaccination confers immunity only after the pustule has passed through all its stages. If the individual has been infected with small-pox prior to vaccination, the variola will develop, but often runs a milder course.

VII. The following is the clinical history of vaccination:

During the first few days after vaccination, hardly any change is noticeable. On the fourth day, a red papule appears on the site of vaccination. On the fifth day, serum accumulates beneath the epidermis of the papule, so that a small vesicle appears. On the sixth day, this increases in size, its contents become cloudy, and its periphery is surrounded by a red zone. On the seventh day, the vesicle is converted into a pustule; its contents are purulent, its red border slightly infiltrated, around it is a hyperæmic zone shading gradually into the healthy parts. The pustule increases in size in the next few days, and attains its greatest development on the tenth day. On the twelfth day, the contents of the pustule begin to dry, and a crust forms which falls off on the twenty-first day. At first its site is occupied by a lattice-shaped red cicatrix, which gradually becomes white, and persists for life.

General symptoms are usually absent. If careful measurements are made, the bodily temperature will be found slightly elevated during the first three days (prodromal fever) and from the seventh to ninth days (suppurative fever). If the temperature rises to 40° C. or more, other febrile symptoms also develop: increased thirst, anorexia, a tearful mood, restless sleep, and even convulsions and delirium.

The anatomical characteristics of vaccine and variola efflorescences are, in the main, identical.

VIII. The pathological accidents of vaccination may be local or general.

In a very few cases death has resulted from hemorrhage from the site of hemorrhage. Strongmeyer and Henoch observed this in a hæmophilic child, Polk in a leukæmic child. As a rule, however, vaccination is well tolerated in hæmophilia.

A painful furuncle, attended with febrile movements, sometimes develops at the site of vaccination, especially if the incision has been made too deep.

Vaccine ulcer is the term applied to those cases in which the pustule develops normally until the tenth day, after which it ruptures and discloses a painful ulcer. This is accompanied by general febrile disturbances. It is relatively frequent after vaccination with primary bovine lymph.

The term vesicular pocks is applied to those cases in which large vesicles with watery contents are formed. Thin crusts are produced, and fall off without leaving a cicatrix.

In eczema pocks, a series of vesicles form around the site of vaccination, and terminate in a weeping eruption. This is observed with relative frequency in anæmic, rachitic, and scrofulous children, especially if they have suffered previously from eczema.

In two cases, Bednar described gangrene of the site of vaccination, and death from collapse.

Vaccination erysipelas may develop early or late. The former appears two or three days after vaccination, and is much more dangerous than the late form, which appears from the tenth to twenty-first days. The erysipelas is the result of the wound, and extends over the arm, and even the larger portion of the trunk. It is often fatal. Its production is favored by uncleanliness in vaccination, vaccination in the hot months, the epidemic occurrence of vaccination erysipelas, the removal of lymph from pocks which are surrounded by a broad zone of inflammation, or

from erysipelatous children, and vaccination with primary bovine lymph. If several cases of erysipelas develop, vaccination should be discontinued for some time. The treatment is similar to that of other forms of erysipelas.

Vaccination is sometimes followed by the appearance of pock-like vesicles over the entire body, but this seems to be the result of accidental combination with varicella.

Roseolar patches which often last only a few hours, and sometimes appear from the third to eighteenth days after the operation, are known as vaccination roseola.

The adjacent lymphatic glands are often tender, but in exceptional cases they undergo suppuration, or become swollen in distant parts, or the salivary glands enlarge. Lymphangoitis is occasionally observed. Bednar states that he has observed peritonitis after vaccination in several cases.

10. *Varicella. Chicken-pox.*

I. Etiology.—Chicken-pox is a disease of childhood, and occurs so rarely in adults that it must arouse the suspicion of varioloid. The susceptibility to the disease generally disappears at the age of ten years.

The youngest case was observed by Senator in a child æt. 11 days. Among 584 cases collected by Baader,

382	occurred	from	1– 5 years.
191	"	"	6–10 years.
7	"	"	11–15 years.
2	"	"	16–20 years.
2 (?)	"	"	20–40 years.

Cases of congenital varicella are unknown.

Sex exercises no influence on the disease.

There is no doubt that the disease is contagious. It often occurs endemically in schools, asylums, or families. Sporadic cases are almost always to be found in large cities. At times it appears in epidemics which seem to be independent of meteorological influences. In many cases, the epidemics end in a few weeks, in others they are protracted for several months.

Positive results have been obtained by inoculating healthy children with the contents of varicella vesicles, although some writers obtained negative results. It is probable that infection may also be effected by the expired air.

Tschamer claims that he has obtained, from the urine and dried crusts, fungus cultures which he regards as the virus of the disease. He describes and draws branching threads, which grow smaller at their free ends, and carry gonidia (?).

As a rule, the disease occurs only once in a life-time. Exceptions to this rule are rare.

Epidemics of varicella often follow, precede, or accompany epidemics of measles, scarlatina, variola, or whooping-cough. A child is sometimes attacked at the same time by varicella and measles, scarlatina or whooping-cough. Thomas observed the development of varicella on the second day of a pleuro-pneumonia. The combination of chicken-pox and small-pox is hitherto unknown.

II. Symptoms and Anatomical Changes.—The period of incubation averages thirteen to sixteen days, occasionally it is as short as eight days or prolonged to nineteen days.

Kroenlein describes the following case which illustrates the length of the period of incubation. January 4th, 1884, a child was received into the surgical ward of the Zurich Hospital, and on January 6th, was attacked with varicella. January 20th, at noon, two other children were taken sick, and the first efflorescences appeared on the evening of the same day. In vaccinations, the period of incubation is generally short (about eight days), and in one case, Fleischmann observed the eruption on the second day after inoculation.

A prodromal stage is entirely absent in many cases, but some children suffer from irritability, anorexia, eructations, vomiting, and irregularity in evacuations from the bowels. Canstatt mentions tenesmus of the bladder and pale urine; disturbances of deglutition have also been observed. Delirium and convulsions occur in rare cases. As a rule, the bodily temperature is unchanged; but occasionally it rises to 39° C. or even more. This stage lasts only a day or two, the elevation of temperature only a few hours.

The stage of eruption begins, in rare cases, with a fleeting erythema. The specific eruption appears first in the face, and rapidly extends to the trunk and limbs; the scalp is often affected.

The eruption appears first as red patches, of the size of a lentil to that of a finger-nail, rarely larger. They grow pale on pressure, and are gradually elevated slightly above the level of the skin. In a short time (six to twelve hours) vesicles form in the centre of the patches, and increase to the size of a lentil or pea; in rare cases they may measure four centimetres in diameter. The large vesicles are always isolated. They are generally oval in shape, and, as a rule, do not occupy the entire circumference of the prodromal roseola, so that they are generally surrounded by a red zone (hyperæmia of the cutaneous vessels). The vesicle is situated in the upper layers of the epidermis and is only covered by a thin layer of epidermis. The centre is not infrequently slightly depressed and less transparent, so that, according to my experience, the vesicles may be umbilicated. The contents of the vesicles are clear and watery, but after the second day they become cloudy from the admixture of cellular elements. Vesicles with purulent contents may develop in rare cases and are exactly similar to well-developed pustules of smallpox.

If a vesicle is punctured with a needle, a clear fluid slowly escapes, of an alkaline or neutral reaction, and poor in cells. Hence the vesicle is multilocular. If this were not so, the contents would be discharged at once.

When the vesicles are left to themselves, slight collapse occurs from absorption of the fluid contents, perhaps in part from evaporation, and the top of the vesicle is wrinkled. About the fourth day, the contents dry and form a thin, horn-yellow, or yellowish-gray crust. Two or three days later, this falls off without leaving anything beyond a red pigmented patch which persists for a few days. In rare cases, a few vesicles extend to the deeper layers of the epidermis and even to the superficial layers of the cutis, so that permanent cicatrices are produced.

The desiccation of the vesicles is attended not infrequently with severe itching, so that the children scratch the vesicles and produce excoriations even in their vicinity. Very tense vesicles may burst spontaneously, and dry up after partial discharge of their contents.

The vesicles are generally distributed irregularly; they are sometimes arranged in groups, as in herpes. Their number varies greatly (from ten to eight hundred or even more); they are most abundant upon the

trunk. Confluence of adjacent vesicles occurs very rarely. The patches and vesicles appear by fits and starts in the course of a few days. Towards the end of the disease, roseola alone may appear.

It is said that the vesicles sometimes have bloody contents, and occasionally they contain air bubbles, as the result of rupture of the epidermis. Crocker states that in children with a tubercular predisposition, varicella is apt to be attended with extensive gangrene of the skin, which is relatively often fatal; according to Hutchinson, this may lead to loss of sight from purulent irido-choroiditis. In a few cases the eruption is abortive, and roseola alone develops.

In rare instances, the eruption appears on the mucous membranes (enanthem), most frequently on the hard and soft palate, also on the tongue, cheeks, lips, nose, and conjunctiva. On the palate, the vesicles have a characteristic shape; on the remainder of the buccal mucous membrane, they generally burst rapidly and leave a shallow ulcer with red borders. The vesicles have also been observed upon the prepuce and labia, where they give rise to a sensation of burning during urination. According to Comby, the enanthem sometimes precedes the exanthem.

In not a few cases, the disease consists merely of the eruption. In others, moderate fever, with morning remissions and evening exacerbations, appears during the first few days. Considerable rise of temperature (to 41° C.) occurs occasionally, and may even be associated with delirium and convulsions.

Mild pharyngitis is frequent; enlargement of the submaxillary and cervical lymphatic glands has also been described. Dry bronchitis is frequent and is easily recognized by the sonorous and sibilant râles.

The disease almost always runs a benign course. Its average duration is one to two weeks; occasionally it is protracted for six weeks. Relapses are occasionally observed.

Erysipelas, in a few cases peritonitis, and otitis have been observed as complications.

Prolonged pallor and chronic feebleness are sometimes left over as a sequel. Pemphigus and urticaria have been observed as sequelæ. In four cases, Henoch observed acute nephritis one to two weeks after recovery from the disease, one case terminating fatally from œdema of the lungs.

III. Diagnosis.—The disease is distinguished from pemphigus by the fact that, in the latter, larger vesicles are produced and run a slower course. In miliaria, there has been preceding diaphoresis, uncovered parts of skin remain free from vesicles, the vesicular contents are acid, and the vesicles disappear very rapidly. In herpes, the vesicles are always arranged in groups. Eczema vesiculosum is associated with violent itching, and the integument between the vesicles is almost always inflamed. In adults, the disease must be distinguished from the syphilitic eruption known as varicella syphilitica; in these cases, other syphilitic changes are present on the skin, mucous membranes, and genitals. Variola is differentiated by the severe prodromal symptoms, and by the fact that vesicles form upon small papules.

IV. Prognosis and Treatment.—The prognosis is almost always favorable, and treatment is generally unnecessary. If there is no fever, the patient need not take to bed, but it is well to be careful in diet, and to guard against taking cold. Exfoliation of the crusts may be accelerated, and the itching diminished by baths at 28° R. In other respects, purely symptomatic treatment.

The spread of the disease can only be prevented by strict isolation, but, on account of its innocuous character, this is rarely necessary.

PART II.

INFECTIOUS DISEASES IN WHICH THE MOTOR APPARATUS (JOINTS OR MUSCLES) IS CHIEFLY INVOLVED.

1. *Acute Articular Rheumatism.*

(*Polyarthritis acuta.*)

I. ETIOLOGY.—The view that this is an infectious disease, produced by specific bacteria, is gradually gaining ground. In our opinion, the term articular rheumatism should be discarded, and the disease should be called infectious arthritis or polyarthritis.

In furnishing grounds for our view as to the infectious nature of articular rheumatism, we need not rely upon Pocock's case, in which a pregnant woman suffering from the disease gave birth to a child which also presented evidences of articular rheumatism; nor upon that of Thoresen, who claims to have observed the spread of the disease by personal contact. On the other hand, I would call attention to the frequent occurrence of the disease in epidemics. In Zurich, I have often noticed that a large number of cases entered the hospital in a few days, so that I have repeatedly had more than a dozen cases in my wards at one time. These epidemics are more frequent in the cold, changeable weather of winter and spring. As is true of other infectious diseases, the character of the individual epidemics varies greatly, but cases occurring during the same epidemic present great similarity to one another.

Edlefsen found that, in Kiel, acute articular rheumatism is a "house disease," like fibrinous pneumonia and typhoid fever, so that, for example, 728 cases occurred in 492 houses.

This writer also found that the frequency of the disease does not depend upon the temperature or variability of the weather, but upon the amount of rain-fall, increasing with diminished rain-fall, diminishing with increased rain-fall.

According to Thoresen, the disease does not flourish above certain levels. Its proper habitat is the temperate zone, especially on the sea-coasts.

Another circumstance indicative of its infectious nature is its intimate relation to other infectious diseases, especially endocarditis and meningitis. Autopsies in cases of this disease also produce the impression of an infectious process; hemorrhages into various organs, cloudy swelling of the heart, liver, and kidneys, soft large spleen, etc.

The majority of patients, it is true, state that the disease is brought on by a cold, but, on careful examination, there is only a very small proportion of cases in which this is probable. Not that we deny the injurious effects of a cold, but, in our opinion, this is secondary, and merely preparatory for the bacteria.

Some patients mention bodily or mental exertion as the cause of the disease.

Heredity sometimes appears to play a certain part. Perhaps, in such cases, the power of resistance of the joints is diminished.

Males are attacked more frequently than females. It is also more common in those who work a good deal in the open air.

The disease occurs most frequently between the ages of 15 and 30 years. It is rare in old age and childhood, but has been observed at birth, or a few days after birth. It presents a marked tendency to relapses.

Many distinguish primary and secondary articular rheumatism. The former occurs independently, the latter follows other infectious diseases (scarlatina, gonorrhœa, diphtheria, syphilis, dysentery, typhoid and relapsing fever, erysipelas, puerperal fever, erythema nodosum). In our opinion, however, we have to deal, in secondary rheumatism, with true metastases.

II. Symptoms.—Prodromata are absent in the majority of cases. In rare cases, the patients suffer from general malaise and wandering pains in the limbs for two or three days previous to an attack.

As a rule, the disease begins suddenly with a chill or repeated chilly sensations. An irregular type of fever sets in, rarely exceeding 40° C. The frequency of the pulse and respirations is increased. The tongue is coated, the appetite lost, thirst increased. The bowels are constipated. The urine is scanty, dark-red, often deposits a sediment of urates, and its specific gravity is increased. It is often extremely acid and not infrequently contains small amounts of albumin.

The amount of urea and uric acid is increased as in febrile conditions generally. Jaksch found peptone in the urine in twelve cases, but only after the swelling of the joints was absorbed. It is probably the result of the absorption by the blood of the exudation cells in the inflamed joints.

The skin is generally covered with profuse, sour-smelling perspiration, which often gives rise to the development of miliaria.

Rapid diminution of the red blood-globules and increase of the white globules have been observed. Salomon did not succeed in finding lactic acid in the blood.

Changes in the joints appear almost simultaneously with the onset of the fever. The large joints (knee, ankle, shoulder, wrist, and elbow) are attacked most frequently, but the smaller ones (fingers and toes) are also often involved. The symptoms often begin in one or a few joints, disappear in three or four days, and then re-appear in other joints. The change sometimes occurs in the course of a single night. Occasionally almost all the joints are attacked, even those of the maxilla, vertebræ, the sterno-clavicular joint, and the synchondroses of the ribs, symphysis, and ileo-sacral articulation. Painful affection of the joints of the arytænoid cartilages has also been described in a few cases.

The diseased joints are thickened and swollen; the overlying skin is red, smooth, and shining. It feels hot, and a more or less distinct groove is left after pressure. In fact, the visible swelling of the joints is the result, not so much of an abundant exudation into the joint cavity, as of œdema of the surrounding soft parts. The slightest movement of the parts is attended with the most violent pain. The patients keep the joints slightly flexed, and if the disease is extensive, are rendered helpless. The situation is especially distressing if the joints of rotation and flexion of the head and of the jaws are also attacked.

Creaking is sometimes felt on passive movement of the joints. This

does not always originate in the joint cavity, but results occasionally from inflammation of adjacent sheaths of tendons.

Drosdoff states that, in examination with moist electrodes, the electrocutaneous sensibility over the affected joints is diminished or even abolished. Abramowski found the sensibility increased when dry electrodes were used. Drosdoff observed diminished pressure sensibility, and increased temperature and tactile sensibility over the inflamed joints. The local rise of temperature varies from 2 to 3° C.

The duration of the disease varies from a few days to four, eight, or twelve weeks. There are frequent remissions and exacerbations, the latter being produced especially by too early or incautious use of the joints. The disease may confine itself to a certain joint in which it continues with great obstinacy. The longer it lasts the less marked the fever, cutaneous and urinary changes become. Recovery generally occurs gradually, rarely in a sort of crisis. The epidermis over the swollen joints often becomes wrinkled, and desquamates actively.

Attention has been called to latent articular rheumatism which occurs as neuralgia, often periodical, usually of the trigeminus; it is associated with endocarditis, and leaves the joints free, but rapidly disappears under the use of salicylic acid.

Complications are remarkably frequent in this disease.

Endocarditis often develops, sometimes even the ulcerative form. It is so much more apt to occur the greater the number of joints affected (vide Vol. I., page 69).

Pericarditis is somewhat rarer (endocarditis in about 20%, pericarditis in 14% of the cases). Both diseases are not infrequently combined.

Dilatation of the right ventricle is often observed, and systolic febrile murmurs are frequently audible. The heart muscle is sometimes subject to embolism, secondary to endocarditis (embolic myocarditis). Some authors also assume the development of non-embolic myocarditis. Others speak of rheumatism of the heart, to which they attribute sudden, sometimes fatal attacks of cardiac pain and heart failure.

Cerebral complications are the result, in some cases, of the high fever which gives rise to disturbance of consciousness and delirium; or, the fever rises so rapidly that death occurs from hyperpyrexia. The temperature sometimes rises above 43° C., and may even continue to rise for a short time after death. Meningitic symptoms may appear, although the autopsy may show merely congestion, hemorrhages, or œdema of the meninges. Cerebral embolism may give rise to paralysis and aphasia. The cerebral symptoms are sometimes the result of uræmic poisoning. Psychopathic conditions have often been observed as sequelæ. All the symptoms mentioned were formerly called cerebral rheumatism.

The complications sometimes start in the joints themselves. The inflammation may become purulent, and terminate in rupture, pyæmia, or ankylosis. Abscesses of the muscles have been observed in a few cases. Myalgia is more common, and may occur in the muscles adjacent to the inflamed joints or in remote parts.

Roseola, urticaria, erythema, facial herpes, erysipelas, and gangrene of the skin have been observed. Purpura and ecchymoses have also been described; upon the latter are sometimes found vesicles with serous, sero-purulent, or sanguinolent contents. Hauff observed herpes zoster at the level of the lower angle of the scapula. Papules some-

times form upon the skin, especially of the forehead and occiput, and disappear with the joint changes.

Irido-choroiditis and cyclitis have been observed in a few cases; paralysis of the motor oculi occurred in one case, but was probably associated with meningitic changes.

Catarrhal angina may appear as a prodromal symptom, or during the later course of the disease.

Bronchitis is of very common occurrence; pleurisy is not infrequent; it is usually unilateral, more rarely bilateral, and occasionally associated with endocarditis and pericarditis. Fibrinous pneumonia has also been observed.

Peritonitis is rare. Acute nephritis and hæmaturia develop at times; anuria may be produced, and may lead to uræmic symptoms. Hæmaturia is sometimes the result of renal embolism, not of acute nephritis. Oppert described hemorrhages from the intestines and uterus.

An unfavorable termination in acute articular rheumatism is always the result of complications, such as rapid rise of temperature, pulmonary embolism, meningitis, pericarditis, heart failure, pyæmia, etc.

Many complications pass directly into sequelæ, for example, ankylosis, which is sometimes followed very rapidly by muscular atrophy; paraplegia and paralysis of the bladder from implication of the spinal cord; hemiplegia and monoplegia from cerebral disease; valvular lesions, etc. Chronic nephritis sometimes develops. Chorea and psychoses merit special attention. Both begin not infrequently during the course of the disease as a complication, extend beyond the primary disease, and persist as sequelæ. Chorea is more frequent in childhood, psychopathy at a later period.

In 1874, Simon collected 61 cases of psychopathy complicating rheumatism. He distinguishes three forms, viz., melancholia cum stupore, recurrent insanity, and imbecility. Maniacal attacks are rare. Relapses of the joint affection are apt to be attended with relapse of the psychopathic, more rarely by recovery of the psychopathic, condition. Recovery generally occurs in two weeks to four months. The insanity seems to result from anæmic changes dependent on complicating heart affections (?).

III. Anatomical Changes.—Little is known concerning the anatomical changes of acute articular rheumatism, because the majority of patients recover. In one of my patients, in whom death resulted from hyperpyrexia, hemorrhages were found in the mediastinum, epicardium, pleura, spleen, beneath the peritoneum covering the intestines, and into the meninges; also parenchymatous hemorrhages into the heart, liver, and kidneys; in addition, cloudy swelling of the heart, liver, and kidneys. Spleen large and soft.

The joints sometimes contain very little fluid; it has probably disappeared in part after death. In other cases, the contents are abundant, flocculent, cloudy, occasionally purulent. The synovial membrane, cartilages, and even the ends of the bones are injected and sometimes contain hemorrhages; the cartilages are occasionally eroded.

The microscope shows proliferation of the cartilage capsules and cells. The fluid exudation contains pus-corpuscles, large cells with several nuclei, and granulo-fatty cells. The flocculi consist of fibrin and mucin.

Fibrinous deposits are said to have been observed on the inner surface of the joints (arthromeningitis crouposa).

IV. Diagnosis.—As a rule, the diagnosis is easy. The disease is distinguished from gout by the fact that the latter generally runs an apyrexial course, and affects the great toe joint. In pyæmic joint diseases, the articular changes are secondary.

V. Prognosis.—The prognosis is good, inasmuch as there is rarely (in hardly 3% of the cases) any immediate danger to life. It may be rendered unfavorable by the complications and sequelæ.

VI. Treatment.—The patient's room should be large, well aired, and kept constantly at 15° R.; if possible, it should contain two adjacent beds, one for the day, the other for the night. Lemonade may be taken as a drink. If fever is present, the patient should take only fluid food.

Among medicinal agents, salicylic acid has displaced almost all others. This drug, or salicylate of soda should be given in doses of gr. vij. every hour until tinnitus aurium is produced. The remedy is again given after the tinnitus has ceased. In many cases, the pains subside very rapidly and disappear within twelve hours. The effects are apt to be so much more prompt the more acute the symptoms, the higher the fever, the greater the number of joints affected, and the more marked the inflammation in them. When the pains have subsided—and the swelling of the joints usually disappears with surprising rapidity at the same time—the salicylic acid should be given every two hours for the next two days, then every three and four hours. If the treatment is not continued for some time, relapses occur not infrequently.

It may happen, however, that one or another joint remains swollen and painful. In such cases, I have obtained good effects from local warm baths containing a pound of salt (thirty minutes' duration, 30° R.). The application of a plaster of Paris bandage may also cause rapid relief of the swelling and pain, but this is sometimes followed by rapid muscular atrophy, requiring the subsequent application of the faradic current.

If salicylic acid proves useless, we may warmly recommend fixation of the joints by splints or bandages, but this plan meets with practical difficulties, if many joints are attacked.

We have often obtained good effects from Davies' plan of treatment. A fly blister (if the joint is large, two blisters) is placed upon the affected joint, the blister opened, the surface covered with carbolized oil, and then inclosed in salicylated wadding. Poisoning with cantharides occurs very rarely.

We may mention the following other methods of treatment: *a.* Derivatives: leeches, cups, moxa, actual cautery, alcoholic inunctions, veratrine ointment, ichthyol ointment (15 : 50), nitrate of silver, faradic current. *b.* Antiphlogistics: ice-bags, ether spray, elayl chloride. *c.* Narcotic inunctions with chloroform, chloroform liniment, ointments of belladonna, opium, etc. *d.* Subcutaneous injections of morphine or carbolic acid (one to three per cent). *e.* Absorbents: tincture of iodine, iodoform ointment. *f.* Antirheumatics: aconite, colchicum, potassium iodide. *g.* Antipyretics: digitalis, tartar emetic, veratrine, quinine, benzoic acid, salicin, antipyrin, kairin, etc. *h.* Diaphoretics: pilocarpine, hot-air chamber. *i.* Laxatives. *k.* Mercurials (internally and externally). *l.* Alkalies: potassium nitrite, sodium nitrite, potassium carbonate, sodium bicarbonate. *m.* Astringents: acetate of lead, ergotin. *n.* Narcotics, opium, morphine, chloral hydrate, ammonium bromide, potassium cyanide. *o.* Specifics: propylamin (useless in many of my cases), tincture cynarne, tincture guaiac, permanganate of potash.

Iodine and iron may be employed in persistent anæmia.

Complications must be treated according to general principles. In hyperpyrexia we should use protracted lukewarm baths (26° R., thirty minutes' duration) and large doses of antipyrin (3 i.–iss. by enema).

2. *Chronic Articular Rheumatism.*

(*Chronic Polyarthritis.*)

I. Etiology.—In many cases, this is a sequel of the acute form. But the disease may also be chronic from the start, and is then generally attributed to colds, to repeated wetting, and living in damp rooms. As a rule, the patients are past the age of 40 years. Heredity is sometimes said to play its part in the etiology.

II. Symptoms.—The chief symptom is pain in the joints, which develops spontaneously, on pressure, or on movement. The joints are often, though not always, swollen, and at times the overlying skin is red and œdematous. The ankles, knees, shoulders, elbows, and wrists are attacked most frequently; but similar changes may also appear in the fingers and toes.

After the inflammation has lasted for some time, creaking of the joints may develop, motion becomes difficult, and even ankylosis may be produced. The capsule, ends of the bones, and the inserted fasciæ may be permanently thickened.

Fever is absent. As a general thing, there are no complications on the part of the heart or other organs.

The disease lasts many weeks, months, years, or even a lifetime. Remissions and exacerbations are frequent; the latter occur particularly during changeable weather.

Special morbid causes occasionally produce exacerbations of such a severe character that the symptoms of acute articular rheumatism are produced.

The complications and sequelæ consist of the previously mentioned deformities of the joints and ankyloses. These are sometimes followed by very rapid muscular atrophy which cannot be attributed entirely to disuse.

III. Anatomical Changes.—These consist of thickening of the synovial membrane, the villi and capsule of the joint, sometimes of adhesions within the joint cavity. The articular fluid is usually scanty. Erosion of the cartilages may occur as the result of fatty, fibrous, and mucoid degeneration of the cartilaginous tissue.

IV. Diagnosis.—The diagnosis is evident from the symptoms. The disease is distinguished from gout by the fact that it runs a more gradual course, and that the great toe is not attacked with special frequency or severity. The differential diagnosis from arthritis deformans is more difficult; chronic rheumatism is often preceded by acute attacks, and inflammatory changes prepare the way for deformities of the joints.

V. Prognosis.—While there is no danger to life, the disease can only be improved, not cured, by medical interference.

VI. Treatment.—Trial may be made of salicylic acid or salicylate of soda, but the effects are uncertain, and can only be looked for if large doses are continued for a sufficiently long time (gr. xv. every hour until tinnitus aurium is produced repeatedly). Potassium iodide (gr. xv. t. i. d.) may be given; also tincture of aconite, colchicum, or other remedies employed in acute articular rheumatism.

During the summer the patient may be recommended to take a course of indifferent thermal baths, sodium chloride, or sulphur baths. Benefit may also be derived from mud baths, Russian or Turkish baths, and cold-water cures. The poor must be satisfied with ordinary warm baths (30° R.), perhaps, with the addition of salt or sulphur. Trial may also be made of sweat cures.

The local measures include leeches, cups, alcoholic and narcotic inunctions, subcutaneous injections of morphine or carbolic acid, massage, and electricity. Seeligmueller recommends the faradic brush (strong current, the brush as the negative pole).

3. *Muscular Rheumatism.*

I. Etiology and Symptoms.—Muscular rheumatism is characterized by pain in the muscles, which appears spontaneously or is produced by pressure. The pains may be confined to one muscle, or they jump to various muscles. As a rule, the disease is apyrexial, and elevation of temperature only occurs when many muscles are attacked. The usefulness of the limbs may be considerably impaired. Pain and difficulty in respiration, cyanosis and dyspnœa may be produced by rheumatism of the chest muscles. In one of my cases the rheumatism of all the muscles of the back was so violent as to give rise to marked opisthotonos. When the neck muscles are attacked, the head is held stiff; if the affection is unilateral, rheumatic torticollis is produced. We must avoid mistaking rheumatism of the abdominal muscles for peritonitis.

The disease may be acute or chronic, the former lasting a few days, the latter lasting weeks and months, and often presenting remissions and exacerbations. Muscular contractures or fibrous thickenings sometimes develop.

Endocarditis and myocarditis have been observed in a few cases as complications of muscular rheumatism.

The disease generally develops beyond the age of thirty years, and is attributed, as a rule, to colds, living in damp rooms, etc., sometimes to hereditary influences.

II. Anatomical Changes are not discoverable. The diagnosis is easy, the prognosis favorable, except in old cases. The treatment is similar to that of chronic articular rheumatism.

PART III.

INFECTIOUS DISEASES IN WHICH THE BLOOD AND HEMATOPOIETIC ORGANS ARE CHIEFLY INVOLVED.

1. *Relapsing Fever.*

(*Recurrent Typhus.*)

I. Etiology.—The mode of infection and spread of relapsing fever is similar to that of typhus fever. As a rule, the disease is contracted by direct personal contact, more rarely through the medium of intermediate persons, still more rarely by various utensils, clothing, or bedding. In hospitals, the nurses, laundresses, and physicians are often attacked, and the disease sometimes spreads to adjacent beds, when the patients are placed in general wards.

The disease occurs chiefly among poor people and tramps. An epidemic often spreads from a single imported case, especially in miserable lodging-houses and prisons. In our opinion, its autochthonous development is impossible.

In Ireland and Russian Poland the disease occurs endemically, and Jewish immigrants from the latter country have started a number of epidemics in Great Britain. Irish immigrants, in like manner, have imported the disease into England and America.

Repeated importation into a city or State may finally convert the latter into an endemic site of the disease.

As in the case of typhus, the outbreak of epidemics of relapsing fever is favored by failure of crops, famine, and war, but is uninfluenced by the character of the soil, climate, temperature, drinking-water, etc.

The majority of cases occur between the fifteenth and twenty-fifth years, although the disease is not rare in children, especially from the fifth to tenth years. As a rule, the disease does not occur during the first year of life, but Albrecht showed that it sometimes attacks the fœtus in utero. It is rare beyond the age of forty-five years.

Sex, occupation, and constitution exercise no noteworthy influence on its frequency. Pregnant women have been attacked in a number of instances. Premature delivery is generally produced, the child being still-born or living only a few days.

The virus is undoubtedly contained in the blood, but inoculation is attended with successful results only when the blood is taken from the patient during a febrile seizure. The blood is not infectious during the period of incubation, and also loses this property ten weeks after the last attack of fever.

In Motschutkoffsky's experiments, the blood remained infectious even after being kept for two days in capillary tubes at 10° R., or diluted with an equal amount of a one-tenth per cent solution of hydrochlorate of quinia. The virus was not contained in the saliva, sweat, milk, urine, and excrement.

Inoculations of animals were unsuccessful, except in monkeys. It almost seems as if the animal body possesses the power of modifying the poison. At least Carter states that in monkeys suffering from the disease the spirilli are shorter than in man and present fewer turns.

The physical shape of the virus is unknown. Movable schizomycetes (spirochæte Obermeieri) are always found in the blood during a febrile paroxysm, but they are not the carriers of the infectious matter. The latter must be sought in the spores of the spirochætes. Motschutkoffsky found that the blood remained infectious even after the spirilli were killed by the addition of a solution of quinine (0.1%). Hence it is inferred that the spores are more resistant than the spirilli. It has been supposed that the spores are those small shining granules which are found in the blood (?). Carter believes that the spores develop into spirilli in the walls of the splenic veins.

The corpse retains the infectious power for a short time. Perls was attacked with severe relapsing fever immediately after making an autopsy. Heydenreich found moving spirilli in the blood nine hours after death, the body having been kept at a temperature of 36.8° C.

A single attack generally confers immunity in subsequent epidemics, although a few cases have been reported in which individuals were attacked several times.

Epidemics of relapsing fever are sometimes associated with typhus,

more rarely typhoid fever. The individual is sometimes attacked by relapsing fever, and immediately afterwards by typhus, or vice versa. It has also been found on several occasions that when relapsing fever and typhus were prevalent at the same time, the former attacked mainly the poor, the latter the well-to-do. It has also been observed repeatedly that patients suffering from intermittent fever were attacked by relapsing fever, or that the former developed immediately after the latter.

II. Anatomical Changes.—Rigor mortis develops early and is very prolonged. The skin often has a slightly yellowish color, and may contain petechiæ. In some cases there is intense icterus, and then the internal organs are also jaundiced. The body is not emaciated to any noteworthy extent. As a rule, the muscles are dry and have a deep-red color. Hemorrhagic inflammation and softening of the rectus abdominis are observed occasionally, as in typhoid and typhus fever. Small hemorrhages are often found in the muscles and viscera.

The microscope shows cloudy swelling and fatty degeneration of the muscular fibres and proliferation of the muscle nuclei.

The heart muscle is pale, brittle, and flaccid, and in some cases hardly a muscular fibre can be found which is not in a condition of cloudy swelling and fatty degeneration. The bronchial mucous membrane is almost always swollen, red, and covered with profuse secretion. The bronchial glands are often enlarged and congested.

Atelectasis and hypostasis are commonly found in the lungs.

The spleen is sometimes enlarged to five or six times its normal dimensions. Its capsule is tense, and not infrequently presents recent perisplenitic deposits. The splenic pulp is intensely red and diffluent. The Malpighian corpuscles are enlarged, and visible as gray or yellowish nodules. Their centre is often necrotic or contains an abscess. Larger abscesses also occur with relative frequency, and generally develop from wedge-shaped or simple hemorrhagic infarctions.

Rupture of the spleen has also been described, either as the result of excessive swelling or of previous suppuration (in 5.9 per cent of all cases, according to Petersen). The rupture is said to occur most frequently on the surface directed towards the stomach.

The enlargement of the spleen depends partly on congestion, partly on hyperplasia of the cellular elements. A striking phenomenon is the appearance of large fatty cells, which pass from the spleen into the general circulation, and are also found in the blood of the splenic vein and portal vein.

The changes in the follicles start from the central arteries, in which round cells accumulate and undergo rapid fatty degeneration; this results in the formation of central cavities. Granulo-fatty cells are found in the parenchyma of the splenic follicles, and in the adventitia and muscular coat of the arteries. The endothelium of the splenic veins also undergoes fatty degeneration and desquamation, so that spindle-shaped cells are found in the circulation during life.

It has been suggested that the frequent infarctions may be the result of occlusion of the vessels by masses of spirilli, but this has not been substantiated by microscopic examination. Spirilli have been found in the necrotic splenic follicles, and in the blood of the splenic vessels.

As a rule, the liver is very large, and its cells in a condition of cloudy swelling and fatty degeneration. Infiltration with round cells is noticed along the branches of the portal vein. Recent perihepatitis has been described a number of times. The gall-bladder is often distended, generally with dark-green bile, which may be mixed with shreds of mu-

cus. The mucous membrane of the ductus choledochus is often swollen at its entrance into the duodenum, or is occluded by a plug of mucus.

In the bilious typhoid variety of relapsing fever, the hepatic changes are similar to those of acute yellow atrophy of the liver.

Gastro-intestinal catarrh is frequent, and there may also be bloody suffusions and swelling of the follicular apparatus. The mesenteric glands are not infrequently enlarged.

The kidneys are large and flaccid, and contain hemorrhages. According to Ponfick, these are found particularly in the convoluted tubes and Henle's loops. The epithelium of the tubes is in a condition of cloudy swelling and fatty degeneration. Small hemorrhages and slight inflammations are also noticeable on the mucous membrane of the urinary passages.

Meningeal hemorrhages and œdema of the brain have often been noted.

Fig. 24.

Blood in relapsing fever with spirochæte Obermeieri. Immersion. Enlarged 1150 times.

Ponfick found changes in the medulla of the bones similar to those in the follicles of the spleen; they are manifested macroscopically by branching chalky white lines. As in the spleen, so spots of softening develop in the medulla of the bones, and may produce cysts, abscesses and carious changes in the bones; the medulla also contains many granulo-fatty cells.

III. Symptoms.—The duration of the stage of incubation is five to seven days.

A prodromal stage is absent in many cases. In others, general disturbances (malaise, anorexia, etc.) are felt for a few hours or days.

The disease generally begins with a violent chill or repeated chilly sensations. Very high fever rapidly develops, the patients complain of violent throbbing in the temples, headache, and are often so dizzy that they stagger like a drunken person, and are unable to stand. They complain of pain in the back, especially the loins, and shooting pains in the legs. True neuralgias may also develop. The feeling of prostration is especially pronounced. The conjunctiva is injected, the sclera slightly

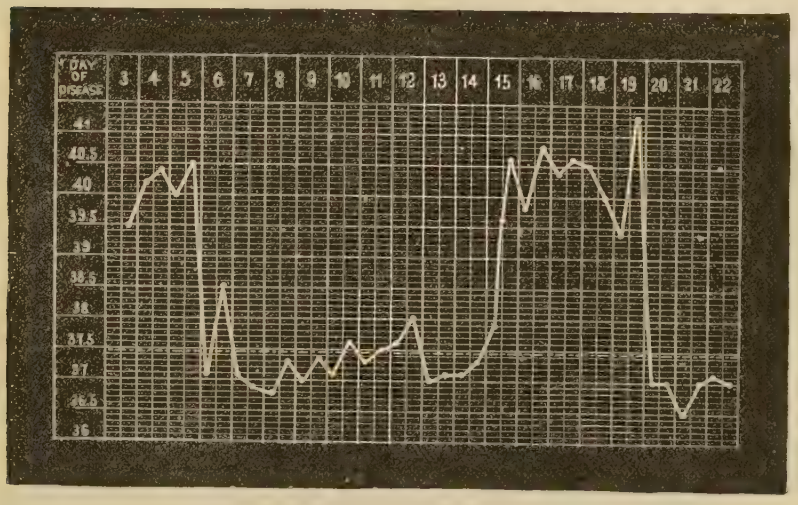
yellow, the face often very pale and almost cachectic in appearance. Some patients experience, at the onset of the disease, a distressing feeling of oppression in the epigastrium, with or without vomiting.

In the further course of the affection, the chief symptoms refer to the blood, bodily temperature, spleen, and liver.

The blood obtained by pricking the finger often has an intense blackish-red color. Under the microscope it is found to contain corkscrew-like, rapidly moving structures (spirochæte Obermeieri, vide Fig. 24). These so-called spirilli are so constant in relapsing fever that the diagnosis cannot be made if they are permanently absent.

Heydenreich noticed that they are sometimes present a few hours before the attack of fever, and when the axillary temperature is below 38° C. As a rule, they occur only with the beginning of the fever, sometimes even several hours later. Towards the crisis their movements become slower, and they usually disappear before the crisis is over. In two cases, however, they were found upon

Fig. 25.



Temperature curve in relapsing fever with one relapse.

the second and third days after the cessation of the fever. With the next attack of fever they reappear in the blood. Their number varies greatly. Motschutkoffsky noticed that they are most abundant before the crisis of a third attack of fever.

In examining a specimen of blood, we should look for any apparently spontaneous movement of red or white blood-globules, which is found not infrequently to be conveyed by spirilli. They often dart rapidly across the field, pushing aside everything that comes in their way. Sometimes they are arranged in coils or one above another.

The spirilli form fine threads, 16–40μ (1μ=0.01 mm.) in length, and with 5–18 corkscrew-like twists. Their forward movement is attended with rotation around the long axis, forward and backward movements in toto, and undulations along their entire length. The more extensive the coagulation of the blood, the slower their movements become. Finally they are surrounded by fine granules into which they appear to dissolve. Their structure is homogeneous; rarely they contain fine granules.

They may be preserved alive for a long time outside of the body. Motschutkoffsky found them alive in the blood at the end of thirty-seven days. Muellendorf preserved them for eight to ten days in capillary tubes. They are very sensitive to reagents, and are killed by everything which alters protoplasm, for

example, distilled water, glycerin, sodium chloride, potassium iodide, permanganate of potash, creasote, sugar, and white of egg. They are more resistant to solutions of strychnine, bicarbonate of soda, salicylic acid, and hydrochloric acid; 0.5 per cent solution of sodium chloride acts like the serum of the blood. In human milk their movements continued eight hours, in perspiration two hours, in saliva one to four hours, in cow's milk one hour; urine and bile exercise a very unfavorable effect. According to Heydenreich, a temperature of 43–46° produces death in one and three-quarters to three and a half hours. At a temperature of 0° they become rigid, but recover if the low temperature has not continued too long. Vapor of chloroform, carbonic acid, oxygen, and the electrical current also produce rapid death. Koch found that, in cultures, the spirilli developed into long threads which were interlaced among one another, but always retained their twisted shape.

The spirilli are found in none of the secretions and excretions of the body.

The white blood-globules are increased in number during the febrile paroxysms. The blood also contains large, granular, in part fatty cells —so-called protoplasm cells—which disclose several nuclei on the addition of acetic acid, and are capable of amœboid movements. These cells are also found in the spleen in which they probably originate. Spindle shaped fatty cells (endothelium of the splenic veins) are also found. A few of the protoplasm cells may also contain one or more vacuoles or red blood-globules. Finally, we may mention the granules—so-called protoplasm granules—which are supposed by some writers to be the germs of the spirilli.

The initial chill is followed by rapid rise of temperature, which soon reaches 40, 41, or even 42° C. The fever generally runs a continued type for five to seven days. Then follows a critical fall of temperature, the patient recovers very rapidly, and remains free from fever for five to seven days. Then follows a relapse with same symptoms as before, and even a third, fourth, or fifth relapse may occur. But the later relapses are usually shorter and less typical. After the disease has run its course, the diagnosis can be made from the temperature curve alone.

In rare cases, the disease terminates with a single paroxysm. If intermittent fever is prevalent at the same time, an intermittent type of fever, with chill and sweat, is sometimes observed at the beginning of the disease. More frequently relapsing fever terminates, as it were, in intermittent fever.

The crisis is sometimes preceded by critical perturbation; in such cases, I have observed a severe chill with relative frequency. The crisis generally occurs at night, and the temperature often falls 5–7° C. in three hours.

The spleen is generally very large, and the patients often complain of pain in the region of the organ. It is said that a systolic blowing murmur is sometimes heard over the spleen. Friedreich noticed that the splenic enlargement appears before the onset of the first paroxysm of fever. After the paroxysm, the size of the organ diminishes considerably, and again enlarges in the next paroxysm.

The liver also increases in size, and is tender on pressure.

All other symptoms are the result, partly of the fever, partly of the infectious process.

The pulse is accelerated (120 to 140 beats a minute) out of proportion to the rise of temperature. It is generally hard and full, rarely dicrotic at the height of the fever, more frequently so after the crisis. Arhythm of the pulse may occur. After the crisis, the frequency of the pulse is sometimes subnormal.

The sensorium is generally unaffected, and delirium is decidedly rare. Many patients complain of obstinate insomnia. There is generally ringing in the ears and difficulty in hearing, partly from tubal catarrh, partly from severe changes in the middle ear. The tongue is covered with a white, yellow, or brown coating, is thickened, and the impressions of the teeth are seen not infrequently upon its edges.

There is often disagreeable fœtor ex ore. The patients complain of a bad taste in the mouth and increased thirst. The appetite is very little diminished in some cases despite the high fever. There is often a complaint of dryness and burning in the nose and throat, and difficulty in deglutition.

The skin often has a light grayish-yellow color. Roseola is often seen upon the abdomen and chest, occasionally herpes labialis, more rarely herpes nasalis or auricularis. Extensive erythema, or petechiæ and urticaria are observed at times. Litten described bluish-red patches, which do not grow pale on pressure, upon the anterior surface of the trunk and thighs. The skin is almost always hot and dry; more rarely the disease begins with sweating. The crisis is always attended with profuse diaphoresis, which often gives rise to miliaria. Hyperalgesia, more rarely analgesia, has been repeatedly described. Desquamation of the skin usually takes place during convalescence, sometimes in larger shreds.

Dry bronchitis is an almost constant symptom. The right side of the heart is not infrequently dilated, and the first sound is often very feeble and indistinct. I have observed vigorous pulsations of the carotid and temporal arteries in several cases.

The abdomen is sometimes distended and tender on pressure. Some patients experience very violent pains in the region of the kidneys.

The urine presents the characteristics of febrile urine: small quantity, dark-red color, very acid reaction, and high specific gravity. At times, there is a temporary increase in the amount of urine excreted. After the febrile paroxysm, the urine again becomes normal. During convalescence, the amount excreted may again become very large (six thousand cubic centimetres). Albuminuria occurs very often during the fever, and the sediment may contain hyaline and granular casts and tubular epithelium; the formed elements may also be present without albuminuria.

So long as the bodily temperature is elevated, the amount of urea is increased; this increase is not so great in the subsequent attacks. According to Bock and Wyss, the amount of uric acid is diminished during the paroxysm; according to other writers, it is increased. During the febrile period, there is also increase of ammonia and sulphuric acid in the urine. The chlorides diminish to a mere trace, and gradually increase in amount during the intermissions. In a diabetic patient suffering from relapsing fever, the sugar in the urine disappeared, according to Semon and Traube, during the febrile period, and the specific gravity of the urine diminished at the same time.

There are often several thin evacuations from the bowels every day, and these often contain a large amount of bile.

Recovery generally takes place very rapidly, but it is so much slower the greater the number of febrile paroxysms. Complications and sequelæ are not uncommon.

In addition to delirium, nervous symptoms sometimes appear in the shape of epileptiform convulsions and trismus. In rare cases, consciousness is impaired to such an extent that urine and fæces are passed invol-

untarily or the bladder becomes distended to the level of the umbilicus. All these symptoms occur during the febrile period. There may be considerable rigidity of the back of the neck, which is not always due to meningitis, but to pain in the muscles of the nape. Violent delirium sometimes appears immediately after the crisis (inanition delirium). Temporary insanity develops occasionally during convalescence. Paralyses are sometimes left over, for example, atrophic paralysis of the arm and ocular paralyses. Griesinger mentions diabetes mellitus as a sequel.

The eye is often attacked by sequelæ, and it seems as if the frequency of such changes varies with the character of the epidemic. They are more frequent in men than in women. Flocculent opacities of the vitreous are observed not infrequently, and may be combined with iritis, irido-choroiditis, and irido-cyclitis. The posterior surface of the cornea sometimes contains fine light dots (Descemetitis) and hypopion. Keratitis may also develop. Retinal hemorrhages, phlyctænular conjunctivitis, transitory amaurosis, and paresis of accommodation have also been observed.

Catarrhal and purulent inflammation of the middle ear has been described in a number of cases.

Stomatitis, pharyngitis, and swelling of the follicles of the tongue have been observed. Purulent parotitis and inflammations of the submaxillary gland sometimes develop as sequelæ. Croupous deposits have been found in the stomach. Bloody or dysenteriform stools may be the result of necrotic or diphtheritic changes in the large intestine.

Similar changes have been found on the mucous membrane of the larynx and bronchi; ulcers like those of typhoid fever have been seen upon the posterior wall of the larynx. Œdema of the glottis also occurs. Bronchitis is sometimes complicated with atelectasis, hypostasis, catarrhal or fibrinous pneumonia, rarely with abscess or gangrene of the lungs. Huff observed hæmoptysis. Pleuritis, pericarditis, and endocarditis occur rarely; inflammations of the serous membranes are sometimes hemorrhagic.

Peritonitis is equally rare. Rupture of the spleen sometimes occurs, and is followed by rapidly fatal peritonitis. Abscess of the spleen is manifested by chills, renewed fever, and sweats; it may perforate into the peritoneum, pleura, lungs, pericardium, muscles of the loins, stomach, or intestines.

Hæmaturia sometimes occurs, and in Leyden's cases the urine contained spirilli which were evidently derived from the blood. Chronic Bright's disease may develop as a sequel.

Pseudo-menstrual discharges of blood from the genitals is observed at times.

Abscesses or furuncles of the skin may occur as sequelæ. In rare cases erysipelas or bed-sores develop. Equally rare is gangrene of the skin, for example, the ears, nose, lips, or scrotum. Arterial thrombi may also give rise to gangrene of the limbs. Pustular, bullous, or lichenoid eruptions, or inflammation and suppuration of the lymphatic glands may develop during convalescence. Desquamation of the skin has been observed in a number of cases.

The patients are sometimes anæmic long after recovery from the disease, and œdema of the skin may develop.

During the febrile paroxysms, the joints sometimes present changes similar to those of acute articular rheumatism. Contractures of the muscles have been described.

Hepatic complications include catarrhal jaundice with its well-known symptoms. In some cases these terminate in the symptomatology of bilious typhoid, which is undoubtedly a variety of relapsing fever, as has been shown by inoculations. In certain epidemics, this form of the disease is frequent. The condition is a combination of relapsing fever and grave jaundice, the latter being hæmatogenous in origin, and resulting from the severe infection. The jaundice becomes very intense, consciousness is clouded, hemorrhages occur into the skin and mucous membranes, and many patients die from collapse in the first attack.

In uncomplicated, moderately severe cases, the duration of relapsing fever varies from four to five weeks. Death may occur in the first attack of fever from excessive rise of temperature, paralysis of the heart, collapse, or cholæmia; it may also be produced by the sequelæ.

IV. Diagnosis.—The diagnosis is rendered certain by the discovery of spirilli and by the characteristic temperature curve.

V. Prognosis.—In uncomplicated cases the prognosis is good, and the mortality often does not exceed two per cent. It is rendered grave by complications, and is very serious in bilious typhoid, in which the mortality may reach sixty per cent.

VI. Treatment.—The prophylactic measures are the same as those employed in typhus fever. This is also true of treatment (vide page 124). In bilious typhoid, Griesinger obtained good effects from large doses of quinine (gr. xxx.). Kairin is said to be useful; otherwise antipyretic treatment produces very little effect. Bogourdow has stated recently that Fowler's solution causes disappearance of the spirilli from the blood and shortens the fever, but other writers have used this remedy without benefit. Oks claims that no relapses occur in 60% of the cases, if calomel is administered.

2. *Malaria.*

I. Etiology.—Malarial diseases are also known as marsh fever, and are especially frequent in marshy regions.

Endemic malaria is often found near the banks of large streams and the shores of lakes.

The sea shore, likewise, is often the site of endemic malaria, for example, the coasts of the North Sea. Those regions are especially dangerous in which the waters of rivers and seas mingle, and the waters become brackish.

Foci of malaria are sometimes formed in an accidental manner, for example, after inundations, or heavy rains followed by a period of dry heat, digging of ditches, draining of swamps, etc. It has also been observed after volcanic eruptions. It also develops when previously cultivated districts are allowed to fall into disuse, or if barren districts again are subjected to cultivation. Malaria has also been known to develop in vessels as the result of stagnation of bilge water.

The conditions necessary to the development of malaria always obtain where decomposition of vegetable matter is associated with a certain degree of moisture in the soil. The richer the superficial strata of the soil in organic matter, and the more porous to moisture, the more favorable are the conditions for the production of the malarial poison. Hence the disease is sometimes found at high levels.

Malarial poisoning is a prototype of miasmatic infection. Whoever enters the malarial region is in danger of infection; as a general thing,

infection does not result from personal contact with diseased individuals. Whether infection occurs through the medium of drinking-water is still an unsettled question. The poison seems to be propagated with greater facility near the surface of the ground.

According to some accounts, the disease may be produced by personal contact. Sawyer states that he visited a district which was entirely free from malaria, was there attacked by malaria which he had contracted in his own home, and at the end of nine days infected his nurse who lived in the non-malarial region. Buchner claims that the perspiration of the patients may convey the disease to those sleeping in the same bed.

Malaria occurs endemically, epidemically, pandemically, or sporadically. In regions in which the disease is endemic, the cases increase in such numbers at times as to constitute an epidemic. The majority of epidemics begin in the spring and autumn. The temperature is also important. The higher it rises, and the more the swamps are dried up, *i. e.*, the more the decomposition in them increases, the greater is the danger of its epidemic spread. In tropical regions, the spread of epidemics is furthered by the rainy season. A certain influence is also exerted by the winds. Geselle observed that the inhabitants of a village lying near a peat bog were attacked by malaria because the village was constantly exposed to the winds sweeping over the bog, while the laborers in the bog itself were not attacked. It has also been found that a wall suffices occasionally to protect large numbers of people.

Now and then the disease has assumed a pandemic character and travelled into regions which had previously escaped.

Sporadic cases are generally imported from malarial districts. They sometimes recover spontaneously after a stay in a non-malarial region. The first signs sometimes appear after removing to a healthy region.

Sleeping on the damp ground and remaining near marshes early in the morning and late at night are said to be especially dangerous.

The susceptibility to the disease is increased by excesses of all kinds and by colds.

Age and sex exert no influence on the danger of infection. Even the new-born may be attacked with malaria, if the mothers suffered from the disease at the time of delivery.

According to some writers, negroes suffer very little from the disease. Goth maintains that the puerperal condition increases the predisposition to malaria. Among forty-six cases, premature delivery occurred in forty-one per cent.; the children weighed, on the average, one-fourth pound less than normal.

A single attack of malaria predisposes to relapses. If the individual is unable to leave a marshy district, the disease is obstinate and not infrequently continues for life.

In the tropics dysentery and malaria are often prevalent at the same time. Epidemics of cholera have been repeatedly known to be preceded by intermittent fever. It has also been found that typhoid fever sometimes precedes or follows malaria. During malarial epidemics, other diseases sometimes assume an intermittent character, for example, Porter reports intermittent hemorrhages from an amputation stump.

At the present time, the malarial poison is supposed to consist of bacteria. Klebs and Tommasi-Crudeli have described rod-shaped structures--bacillus malariæ--which they cultivated and successfully inoculated in rabbits. Gerhardt recently succeeded in producing intermittent fever in healthy individuals by sub-

cutaneous injections of blood which he had taken during the febrile paroxysm from patients suffering from intermittent fever. Dochmann had obtained positive results by inoculation with the contents of herpes vesicles obtained from patients suffering from intermittent fever.

II. Symptoms.—There are several forms of malarial disease which are known as intermittent fever: latent intermittent, pernicious intermittent, remittent and continued fever, and malarial cachexia (primary and secondary).

The stage of incubation varies, as a general thing, from seven to twenty-one days. But it is said that the symptoms sometimes appear within a few hours after infection. Thus, several physicians have reported that, immediately after reaching a malarial region, they were attacked by scratching in the throat and larynx, a feeling of dryness and constriction; and, soon after, by other manifest symptoms of malaria. On the other hand, it is said that individuals are sometimes attacked more than three months after leaving the malarial region.

The outbreak of the disease is sometimes preceded by prodromata: pallor, malaise, chilliness, somnolence or disturbed sleep, gastro-intestinal disturbances (anorexia, foul taste in the mouth, fœtor ex ore, eructations, vomiting, diarrhœa, etc.).

The form of malaria depends partly on the locality. In our latitude, pure intermittent or latent intermittent fever predominates; in the tropics and endemic sites of malaria, remittent and continued fevers and cachectic conditions are prevalent. Pernicious and comatose intermittent fever is also most frequent in tropical regions.

Intermittent fever is the most frequent form of malaria in our climate. It consists of febrile paroxysms, occurring at certain definite periods, with apyrexial intervals.

In many cases, the fever occurs at a certain time of the day, lasts a certain number of hours, and recurs in twenty-four, forty-eight, seventy-two hours, etc. In quotidian intermittent, the fever occurs every day; in tertian intermittent, every other day; in quartan intermittent, every two days. Binz recently described a case in which the fever returned once a week (intermittens octava), and the interval is sometimes said to last thirty days (?).

The successive attacks of fever sometimes recur at a little earlier period, so that a tertian may be converted gradually into a quotidian, etc. The opposite condition is also observed at times. A second attack sometimes occurs before the first has entirely run its course.

Double intermittent fever is a special variety. In double quotidian, two attacks occur daily at certain definite times. In double tertian, an attack occurs daily, but the attacks vary in severity, those occurring on alternate days being alike. In double quartan, an attack occurs on two successive days, then follows a day without fever, then two febrile days, etc.

The temperature curves generally suffice to permit a positive diagnosis (vide Figs. 26–28).

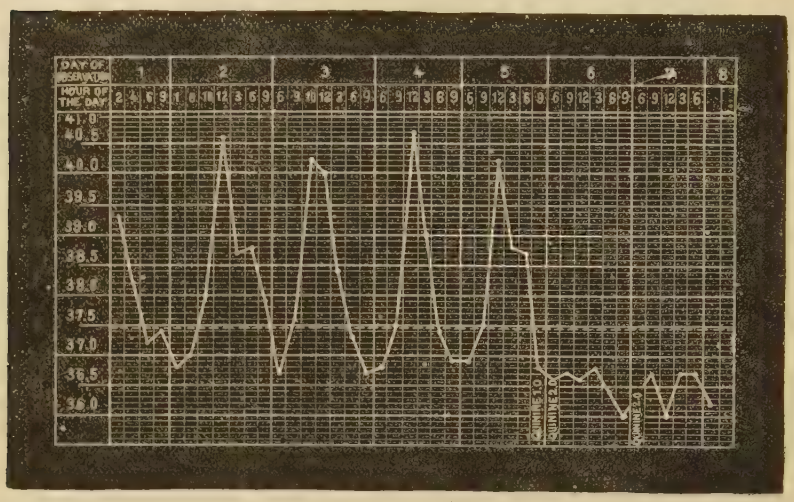
The different types may undergo transformation, so that, for example, the disease begins as a quotidian and is then converted into a tertian.

The paroxysm almost always consists of several stages, viz., the cold, hot, and sweating stage.

In some cases, the stages appear in an inverse order; in others, intervals of hours elapse between the different stages. In erratic intermittent fever, the paroxysms follow no definite order with regard to time.

As a rule, the cold stage begins gradually. The patients feel weak, grow pale, yawn frequently, and stretch themselves. A slight chilly sensation is soon felt along the back, and extends into the limbs. This sensation is gradually intensified into a well-marked chill. The teeth chatter, and the patients sometimes shake with such violence as to move

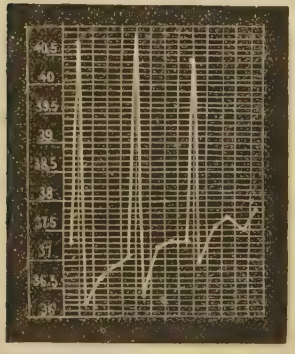
FIG. 26.



Temperature curve in a case of quotidian intermittent.

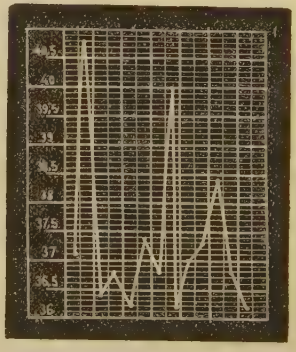
the bed. The skin is pale and feels icy-cold, and its temperature may be 5–7° C. cooler than that of the inside of the body. On pricking the finger, very little or no blood escapes of a deep blackish-red color (con-

FIG. 27.



Temperature curve in tertian intermittent.

FIG. 28.



Temperature curve in quartan intermittent.

traction of the cutaneous vessels, and slowing of the circulation). The skin loses its turgor, the eyes are surrounded by blue rings, the pupils are large, and their reaction slow. Many complain of dizziness and a feeling of syncope, flashes of light before the eyes, and ringing in the ears. The tongue is often coated. Eructations or obstinate vomiting are occasion-

ally observed. The pulse and respirations are accelerated. The patients often, though not always, discharge a large amount of pale, watery urine. The spleen increases in size the more the second stage approaches. There is not infrequently tenderness on pressure over the stomach and liver, or spontaneous pains in the region of the kidneys. The bodily temperature rises during the chill, and reaches its highest point towards the end of the cold stage (even as high as 44° C.).

The cold stage generally lasts one or two hours, not very rarely much longer (six hours or more).

The hot stage begins with a gradually increasing sensation of internal heat which radiates into the periphery. Objectively likewise, the temperature of the skin rises and approaches that of the interior of the body. The latter generally remains the same as at the end of the cold stage, more rarely, it rises a little. The skin becomes turgid, feels dry and burning. The pulse and respirations are still more accelerated. The radial artery is unusually full, and the pulse vigorous. The face is congested, the conjunctiva injected. The dizziness, ringing in the ears, pain and throbbing in the head continue. The temporal arteries are usually very sinuous and pulsate vigorously. The right ventricle is not infrequently dilated, and the first (systolic) sound of the heart often has a blowing quality. A systolic murmur may be heard over the carotids, and a systolic arterial sound over the large peripheral arteries. The physical signs of bronchitis are sometimes present. The spleen increases in size, and continued or systolic vascular murmurs are heard occasionally over the organ. There may also be pain and tenderness over the spleen. The stomach and liver remain sensitive, and the latter is occasionally enlarged. The urine is scanty and saturated (febrile urine).

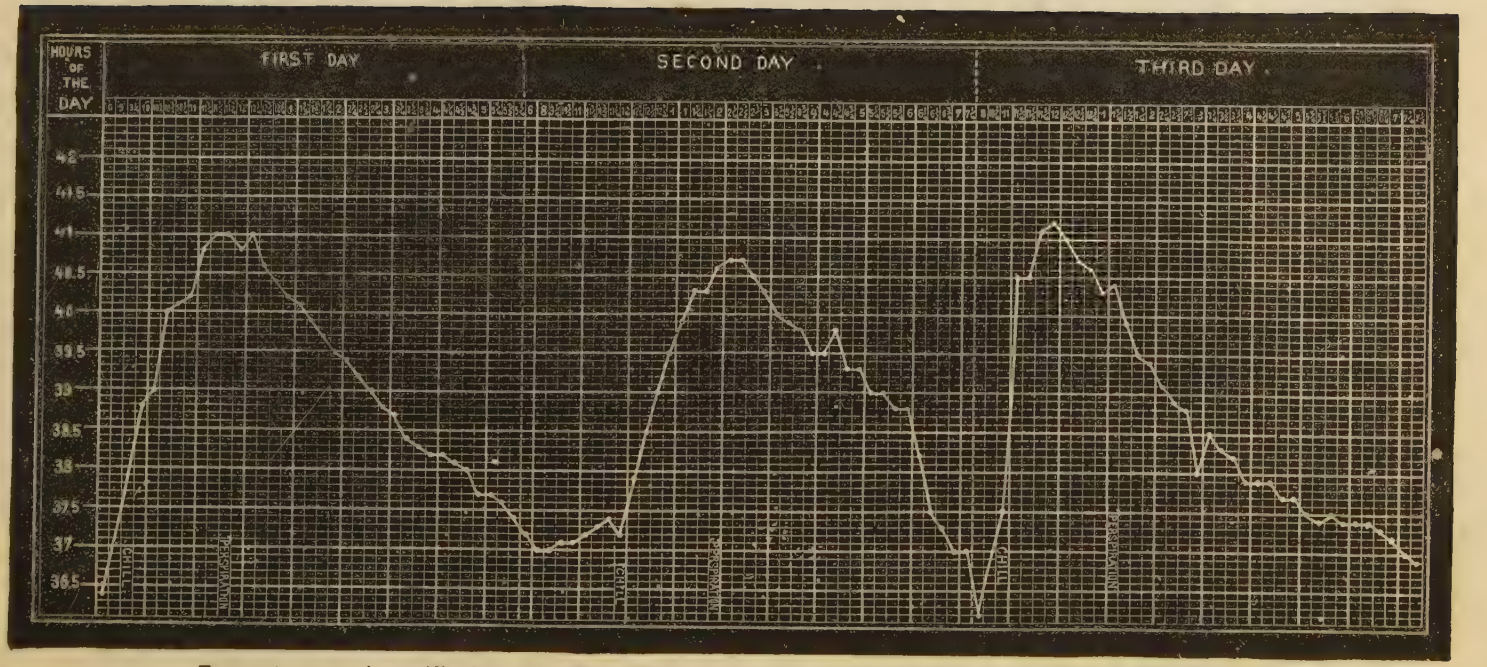
The amount of urea increases considerably during the chill, and reaches its maximum towards the close. Ringer noticed an increase of the urea even before the beginning of the chill. During the hot and sweating stages, the amount of urea gradually diminishes. But A. Fraenkel has shown that a smaller amount is sometimes excreted in the febrile paroxysm than in the apyrexial period, although the production of urea is increased during the former period. If the occurrence of the febrile attack is prevented by the administration of quinine, the increased excretion of urea, nevertheless, occurs at the time when the paroxysm should have appeared.

Uric acid is very little, if at all, increased : the phosphoric acid is increased. While the chlorides diminish in febrile conditions, they increase in intermittent fever, pari passu with the urea.

In one of Riegel's patients, in whom the pulse was not accelerated during the febrile paroxysm, the radial pulse was very dicrotic at this period. Ziehl observed bacilli in the blood, about 4 μ in width, and varying in length from that of a red blood-globule to one-quarter of its length. Sehlen also observed cocci in the blood, partly free, partly in the red globules.

The hot stage lasts three or four hours, sometimes even ten hours or more. When the sweating stage begins, the subjective sensation of heat gradually diminishes in intensity. The integument of the covered portions of the body (beginning in the axillæ) becomes moist, and very large, sour-smelling drops of sweat appear on the forehead and face, and finally on the entire body. The temperature falls, and may even be subnormal at the end of this stage. The pulse becomes full, soft, and slow. The spleen diminishes in size. The urine often, though not constantly, deposits a brick-red sediment of urates. Many patients fall

FIG. 29.



Temperature curve in quotidian intermittent fever. Measurements every fifteen minutes. Duration of disease, four weeks.

into a profound, refreshing sleep from which they awaken with a feeling of relief. As a general thing, the patients recover very rapidly, especially during the first period of the disease.

As a rule, the sweating stage lasts two to four hours. The attacks generally begin between midnight and noon, although there are noteworthy exceptions to this rule. Da Costa Alvarenga reports the case of a woman who was unusually pale, without any apparent cause. Examinations with the thermometer showed that paroxysms of intermittent fever began towards midnight.

Careful examination shows that the rise of temperature during an attack is more rapid than the fall, and the course of both parts of the temperature curve is generally uninterrupted (vide Fig. 29).

The weight of the body sometimes diminishes with striking rapidity. The patient whose temperature curve is shown in Fig. 26 had only two attacks before entrance to the hospital. On admission, he weighed one hundred and fifty pounds; a week later (after five febrile attacks), he weighed only one hundred and forty pounds. Despite recovery, his weight increased very slowly, and, at the end of another week, amounted to one hundred and forty-two pounds.

If the disease is acquired in an endemic site of malaria, it may be very protracted. If the patient passes from a malarial to a healthy region, the disease sometimes recovers spontaneously in one or two weeks. Relapses may be expected so long as the spleen is enlarged.

The complications of simple intermittent fever are not numerous. In children, general convulsions may occur during the cold and hot stage. Gastro-enteric disturbances are also prominent in children. Curtmann mentions as a constant symptom, a leaden gray coating on the tongue from its tip to the circumvallate papillæ. Rupture of the spleen was observed in one case at the onset of the attack, perhaps from distention of the organ with blood on account of the general arterial spasm. Albuminuria, hæmaturia, and hæmatinuria have been noticed in a few cases. Herpes often develops on the lips or other parts of the face; urticaria-like or erythematous eruptions are also frequent.

Among the sequelæ, malarial anæmia occupies a prominent part. Many patients grow pale very rapidly, and the lips and mucous membranes assume a waxy, pale color. Kelsch found that the red and white blood-globules, particularly the latter, diminish considerably in number, especially at the beginning of the disease. Some of the red globules are unusually large. The amount of hæmoglobin sinks to one-eighth the normal. The diminution in the number of red globules is so much greater the larger the size of the spleen. After the attack, the white globules increase in number, and numerous elementary granules are often found in the blood. Melanæmia is one of the sequelæ (vide page 11), and leukæmia may also be associated with intermittent fever. Endocarditis, ulcerative endocarditis, and myocarditis are occasionally observed. Chronic interstitial changes sometimes develop gradually in the liver and kidneys, and there may be extensive waxy degeneration. Dropsy sometimes develops without albuminuria. Glycosuria has also been observed, and terminates occasionally in diabetes mellitus. Parotitis, noma, multiple hemorrhages, chronic dyspepsia, and psychopathies must also be mentioned among the sequelæ.

Latent intermittent fever is characterized by functional disturbances of certain organs, which occur at regular intervals, and are often cured by quinine. They are often preceded by malaise, slight chilliness, and

elevation of the temperature of the skin. The spleen is often, though not always, enlarged.

The disease occurs most frequently as intermittent neuralgia, especially of the supraorbital nerve. It also occurs as intercostal, sciatic, and occipital neuralgia, clavus, ciliary neuralgia, mastodynia, and neuralgia of the testicle. The pain occasionally passes from one nerve to another.

Intermittent paralyses, spasms, hyperæsthesia, anæsthesia, hysteria, psychopathy, delirium, aphasia, agrypnia, and contractures have also been described.

Hydrops articulorum intermittens and intermittent arthralgia (especially coxalgia) are occasionally observed.

The skin may present local or general intermittent œdema or intermittent eruptions (erythema, erysipelas, urticaria, pemphigus, hemorrhages, even gangrenous changes).

Among other symptoms may be mentioned intermittent deafness, blindness, paralysis of the vocal cords, or periodical attacks of sneezing, coughing, asthma, vomiting, eructations, gastric pain, meteorism, hemorrhages from various organs, painful swelling of the breast and testicles, dysuria, constipation, and diarrhœa.

In some cases these symptoms are preceded or followed by manifest evidences of malaria.

In pernicious intermittent fever, otherwise mild symptoms of malaria are intensified to a dangerous degree, or certain unusual organic diseases develop. The danger may simply be owing to the fact that the disease occurs in non-resistant children or old people, or it is owing to the severity of infection. Death occurs often if vigorous treatment is not adopted. The symptoms are not infrequently mild at the start, but pernicious symptoms appear in the subsequent attacks.

The following is a brief resumé of the more frequent varieties:

Algid pernicous intermittent. After a violent chill, the hot stage remains absent, the patients gradually grow colder, the pulse becomes small, and finally death occurs in collapse.

Sweating pernicious intermittent. In the sweating stage, the diaphoresis becomes so excessive that the patients become comatose, and death occurs in collapse unless speedily relieved.

Syncopal pernicious intermittent. Deep and protracted syncope occurs, particularly during the cold stage, and in some cases the patients do not come to again.

Comatose pernicious intermittent. The patients grow more and more comatose, especially during the hot stage; sometimes remain in a state of coma for one or two days, and may die in this condition.

Apoplectic pernicious intermittent. Apoplectic attacks occur, followed by transitory or permanent paralysis.

Epileptic, tetanic, hydrophobic, delirious, and eclamptic pernicious intermittent are sufficiently characterized by their names. A trance condition occasionally develops, and Trousseau states that a patient in this condition had been brought into the dissecting room where signs of life were noticed.

In bronchitic pernicious intermittent, there is violent bronchitis, associated occasionally with dyspnœa and asthma.

Pneumonic pernicious intermittent gives rise to the intermittent development of pneumonia during the febrile paroxysms; pleuritic and pericarditic pernicious intermittent has also been described.

Cardiac pernicious intermittent produces dangerous attacks of palpitation and heart failure, often associated with violent pains in the region of the heart.

Finally, we may mention cardialgic pernicious intermittent (severe intermittent gastric pains), choleraic intermittent (diarrhœa, rice-water stools, and collapse), dysenteric intermittent (bloody stools and tenesmus), icteric and peritonitic pernicious intermittent, hemorrhagic intermittent (hemorrhages from the nose, lungs, stomach, intestines, kidneys, genital organs), amaurotic, erysipelatous, and lymphangitic pernicious intermittent.

Remittent and continued fever occurs chiefly in the tropics, and only during very severe epidemics in temperate zones. Constantly increasing anticipation of the paroxysms occasionally converts an intermittent into a remittent or continued fever and vice versa. Gastric symptoms are prominent, likewise enlargement of the spleen, and often of the liver. Icteric symptoms, bloody stools, and hæmatemesis are sometimes so prominent as to rouse the suspicion of yellow fever. In other cases dysenteric symptoms develop, or the patients lie in a typhoid state, and not infrequently die in collapse.

Fiévre bilieuse-hématurique is the term applied to cases in which severe jaundice and hæmaturia occur. Remittent or continued fever may last from several days to a few weeks.

Malarial cachexia develops not uncommonly after all the various forms of the disease. It may also develop primarily, especially in the localities in which malaria is epidemic. The patients have a waxy complexion, suffer from palpitation of the heart, shortness of breath; the heart is dilated, and systolic murmurs are heard over the heart and cervical veins. Coated tongue, anorexia, vomiting, and eructations are often noticed; the spleen and liver are often enlarged.

Diarrhœa is frequent, and may even be dysenteriform. Tremor, chorea, paralysis, spasms, contractures or grave psychopathic conditions are occasionally produced. Borelli described atrophy of the male sexual organs and gradual approximation to the feminine type.

The sequelæ are similar to those of simple intermittent fever.

III. Anatomical Changes.—The splenic enlargement is the result at first, of excessive hyperæmia. Hemorrhagic infarctions are sometimes observed, more rarely abscesses and perisplenitis. At a later period the spleen becomes hard, and hyperplastic changes develop.

The liver may also present diffuse and miliary hyperplastic changes, in addition to melanæmic processes.

At the present time, most writers are inclined to believe that, while the spleen perhaps acts as a sort of reservoir for the malarial poison, the latter chiefly attacks the nervous system, especially the heat-regulating centres. The periodical recurrence of the attacks and the genesis of latent forms of the disease are unexplained.

IV. Diagnosis.—The chief diagnostic features are the periodical occurrence of the symptoms, the opportunity for malarial infection, and the usually prompt effects of quinine.

Simple intermittent fever may be mistaken at times for pyæmia, ulcerative endocarditis, and pulmonary phthisis. In doubtful cases we

must search for wounds, or changes in the lungs and heart (vide Vol. I., page 75).

Remittent and continued fever may be mistaken for grave jaundice, yellow fever, and dysentery.

V. Prognosis.—The prognosis depends partly on the ability of the patient to leave a malarial region. If this cannot be done, cachexia, extensive waxy degeneration, and incurable marasmus often develop. The more marked the intermittent type the better is the prognosis. Pernicious, remittent, and continued fevers are especially dangerous when their nature is unrecognized.

VI. Treatment.—The prophylactic measures are partly general, partly individual in character. Marshy regions should be drained or brought under sufficient water to prevent decomposition at the bottom. Malarial regions are often rendered innocuous by cultivation.

Individuals should avoid a long stay in poisonous districts, especially after sunset, should avoid sleeping on the ground or drinking water. Colds and excesses of all kinds should be guarded against, and commendable habits of the natives should be imitated. The bed-rooms should be on the upper floors of the house, on the sunny side; the windows should be closed at night.

The prophylactic administration of quinine, arsenic, gentian, or strychnine is not very serviceable, because the organism rapidly becomes accustomed to the remedy.

During the cold stage, the patient should be well supplied with blankets; later, the covering should be light. Pieces of ice or carbonated waters may be given in cases of vomiting.

Quinine is the best remedy in all forms of malaria; gr. xv. to xxx. of the hydrochlorate may be given for two or three days about three hours before the expected attack. One-half or one-quarter the amount may then be continued until the enlargement of the spleen has subsided, and the danger of a relapse has passed. If the quinine is vomited, it may be given subcutaneously (quinia hydrochlorate, glycerin. puri, aq. destil., āā. M. D. S., warmed, two syringefuls) or as an enema with some starch and lukewarm water.

Fowler's solution (five to eight drops t. i. d. after meals) is useful in some cases.

After the febrile paroxysms have subsided, the existing anæmia may be treated with iron and arsenic (tinct. ferri chlorid., 3 v.; liq. potass. arsenit., 3 iss.; gtt. xxv. t. i. d. after meals).

A large number of other remedies have been employed, but all are much less reliable than quinine. *a.* Cinchonia, chinidine, chinoidine, cinchonidine; *b.* Tinct. eucalypt. globuli; *c.* Carbolic acid, salicylic acid, resorcin; *d.* Salicin, berberine, lupinin, apiol, picric acid, strychnine, santonine, buxine sulphate; *e.* Potassium bromide, potassium iodide, sodium chloride, nitrate and acetate of potash; *f.* Liq. ferri sesquichlorid., mercurials; *g.* Chloroform; *h.* Gelsemium sempervirens; *i.* Pilocarpine; *j.* Faradization of the spleen and galvanization of the sympathetic. Hearty eating is considered an effective remedy in certain malarial regions.

3. *The Plague.*

I. Etiology.—Epidemics of plague appeared long before the Christian era. In the great epidemics of the Middle Ages, sometimes more than a fourth of the inhabitants fell victims to the disease. Europe has escaped its ravages since the middle of the fourteenth century, but in 1878–79 there was renewed danger of its introduction from the shores of the Volga.

The disease has also become less prevalent in the Orient. India and Anterior Asia are supposed to be its habitat.

If the plague breaks out in places in which it is not epidemic, it is always introduced from without, so that strict quarantine is the best means of prevention. The disease acquires a so much firmer hold the poorer the surroundings and hygienic conditions of the inhabitants. A single attack does not prevent subsequent infection, although later attacks are apt to be less severe. Age and sex exert no influence; the fœtus in utero may be attacked. It has been held that the plague may develop autochthonously, but this is not probable. The majority of writers do not believe that simple contact with a patient will produce infection, but there is no doubt that it is conveyed chiefly by utensils of all kinds.

The nature of the virus is unknown, but it is said that in the recent epidemics in Astrachan, very small shining bodies were found in the blood, perhaps also in the pus of the buboes.

II. Symptoms.—The plague is an acute infectious disease, in which the external and internal lymphatic glands undergo inflammation, with a tendency to suppuration.

The stage of incubation lasts two to seven days, but longer and shorter periods have also been mentioned.

Hirsch describes three forms of the disease.

In the mildest form, the patients sometimes feel well enough to walk about. Prodromata are absent. The disease often begins with a chill. Then headache, vomiting, and constipation set in. At the same time the glands (inguinal, crural, axillary, submaxillary, cervical) become painful and swollen. In three to six days, some of the glands discharge pus, profuse diaphoresis occurs, and then convalescence. The scars of the buboes are always superficial.

In the moderately severe cases, all the symptoms are more marked. The conjunctiva is injected, and subconjunctival hemorrhages are occasionally found. Delirium and unconsciousness frequently occur, and the temperature of the body is increased. Carbuncles and petechiæ appear on the skin. Tongue thickly coated. Then acute buboes; the escape of pus is often looked upon as a favorable sign. Death in four to six days, or recovery in one to three weeks.

In the gravest form, the patients complain of violent terror, but sometimes retain consciousness until death. The disease may run such a rapid course as to prove fatal before the glands enlarge. There is obstinate vomiting with persistent constipation. The excretion of urine sometimes ceases entirely. Then follow symptoms of dissolution of the blood, especially hemorrhages from the skin, stomach, intestines, kidneys, and lungs. Rapid death from collapse.

Convalescence is sometimes protracted for a long time; relapses are not uncommon. Furuncles of the skin and muscles, parotitis, pneumonia, paralyses, insanity, otitis, and dropsy have been mentioned as sequelæ.

III. Anatomical Changes.—The lesions in the lymphatic glands (external and internal) consist of congestion, inflammatory œdema, and hyperplasia of the parenchyma and periglandular connective tissue; hemorrhages into the glands may also occur. Later, circumscribed necrosis and suppuration of the glands. The spleen is always enlarged, the liver and kidneys swollen and in a condition of cloudy swelling. There are numerous hemorrhages in the viscera.

IV. Diagnosis, Prognosis, Treatment.—The diagnosis is not always easy. The disease may be mistaken for typhoid fever, malaria, splenic fever, and syphilis. The prognosis is grave, and the mortality may exceed 90 per cent.

The spread of the disease can only be prevented by the strictest quarantine. The disease itself is treated symptomatically.

PART IV.

INFECTIOUS DISEASES IN WHICH THE RESPIRATORY ORGANS ARE CHIEFLY INVOLVED.

1. *Whooping-Cough, Tussis Convulsiva.*

(*Pertussis.*)

I. Etiology.—Whooping-cough is an infectious and contagious disease. It generally appears in epidemics, and in most cases it can be shown to follow contact with previously attacked individuals.

The disease is not alone conveyed by immediate contact, but also by intermediate persons (nurses, physicians, relatives, etc.), and by inanimate objects.

It is probably infectious in all stages, to the greatest extent in the convulsive stage. The virus is assumed to be contained in the exhalations from the lungs, since mere presence in a patient's room, without direct contact, suffices for infection. The virus is also contained in the sputum which probably retains its infectious properties even in the dry and powdered condition. It is still doubtful whether the infection is purely local and emanates from the respiratory mucous membrane alone, or whether it is general and originates in the blood. The infection of the fœtus by the mother cannot be explained except as the result of blood infection.

In large cities, sporadic cases occur almost constantly. Hence the possibility for the outbreak of an epidemic at any time. This can sometimes be traced to an imported case.

The majority of epidemics occur in the winter and spring. Their duration varies from less than two months to more than a year. In some large cities, they are said to recur at different intervals. The epidemics are sometimes so mild that hardly any fatal cases occur; in others, the mortality may exceed fifteen per cent.

They sometimes occur at the same time as, immediately before or after, epidemics of other infectious diseases. This is true most frequently of measles, more rarely of scarlatina, small-pox, or varicella. Whooping-cough has also been observed in combination with intermittent fever, erysipelas, and herpes zoster. If the whooping-cough preceded the other infectious diseases, it disappears entirely in mild cases, but in severe cases it is merely rendered milder, and grows worse at a later period. It is said that vaccination sometimes exercises a favorable influence on the course of whooping-cough.

The disease is rare in the tropics. Its development is favored by cold, windy, and rapidly changing weather, because this induces catarrh of the air passages.

Some writers maintain that whooping-cough may develop autochthonously as the result of bad sanitary conditions, dentition, helminthiasis, scrofula, rickets, and even imitation, but this opinion is opposed to all modern doctrines.

The nature of the virus is unknown. Successful attempts at inoculation in animals have been reported by some, but denied by other writers.

The susceptibility to the disease depends very materially upon the age of the individual. It is a disease of childhood, and occurs rarely in adults, though even old people have been attacked in rare cases. It is most frequent from the age of seven months to seven years, and is rare in infants before the age of six months. A few cases have been reported in which mothers suffering from whooping-cough bore children who presented the symptoms of the disease at birth.

It has also been held that whooping-cough in the mother during pregnancy confers immunity upon the fœtus after birth, because it has been infected in utero.

The female sex is attacked more frequently than the male sex. This has been attributed to the greater susceptibility of the female sex to spasmodic affections of all kinds, and to the fact that, among adults, females are more exposed to the danger of infection, because they are chiefly concerned in nursing.

Feeble, anæmic, rachitic, and scrofulous children are more frequently attacked because they possess less powers of resistance, and because they often suffer from catarrhs of the respiratory tract which favor the deposit of the virus of pertussis.

The children of the poorer classes are attacked more frequently than those of the well-to-do (overcrowded dwellings, imperfect isolation of the patients, insufficient care, and frequency of catarrhs of the air passages).

Some individuals enjoy permanent immunity, others only temporary immunity. A single attack confers acquired immunity; individuals are rarely attacked several times.

The susceptibility may be occasionally increased accidentally, for example, by catarrh of the air passages, pregnancy, and the puerperal condition.

II. Symptoms.—The stage of incubation lasts about a week, during which time no symptoms are produced. The duration of this period may vary according to the power of resistance of the patient and the virulence of the poison.

The clinical history may be divided into the catarrhal, convulsive, and decreasing stages. The duration of the disease varies from four or six weeks to as many months or more. On the average, the catarrhal stage lasts two to four weeks, the convulsive stage four to six weeks, and the decreasing stage four to six weeks.

The catarrhal stage not infrequently begins with mild general symptoms. The children lose appetite, grow pale, sleep is disturbed, and there is slight fever. The conjunctivæ are injected, there is slight photophobia, and increased secretion of tears. The patients complain of burning and pricking in the nose, frequent sneezing, and increased secretion of nasal mucus. At times, slight burning in the throat and difficulty in deglutition indicate mild pharyngitis. Cough and slight hoarseness appear, associated with a tickling sensation in the larynx and beneath the sternum. The conjunctival and nasal catarrh diminish, while the cough becomes more frequent and severe, finally it becomes spasmodic, and the disease thus passes into the convulsive stage.

The catarrhal stage is sometimes absent, especially in nurslings.

The convulsive stage manifests itself in characteristic paroxysms of

coughing. They begin with a deep, whistling inspiration, followed by uninterrupted, short, expiratory coughs. Sometimes twenty to thirty expiratory coughs follow one another before another long-drawn, whistling inspiration takes place, to be again followed by expiratory coughing. A single paroxysm lasts from one-fourth to one minute, and a series of them may continue from ten to fifteen minutes. At the end of the paroxysm, tough, glassy mucus fills the mouth and pharynx, and is expectorated by a combination of coughing, strangling, and vomiting movements. In infants, this must often be removed from the mouth with the aid of the fingers.

The paroxysms are often provoked by fright, joy, laughter, crying, etc. In very many patients an attack may always be produced by depressing the tongue with a spatula, until gagging movements ensue. Mimicry also plays an important part. If a paroxysm appears in one of a number of children, the rest very often are also attacked in a short time. In other cases, the paroxysm is preceded by the rattling of mucus in the trachea or larynx, and the irritation of the laryngo-tracheal mucus membrane evidently provokes the attack.

Intelligent patients complain not infrequently, before the paroxysm begins, of intolerable tickling in the larynx, trachea, or beneath the sternum. The desire to cough can rarely be repressed by an effort of the will. The children grow more and more terrified, hold fast to a firm object, or to the nurse, and ask that their head be held between the hands.

During the attack, the face presents evidences of increasing venous stasis. It becomes more and more cyanotic, the eyeballs protrude, the lips and cheeks become swollen, the jugulars appear as blue cords as thick as the finger, the face and limbs are bathed in perspiration, the pulse is felt with difficulty, partly on account of the violent concussion of the body, partly because the heart's action intermits and the pulse becomes smaller. There is sometimes involuntary evacuation of urine, flatus, and fæces.

During the expiratory coughing, the thorax is dull on percussion, on account of the unusual compression of the thorax. During the whistling inspiration, the percussion sound becomes very loud. The respiratory murmur is not audible during the long-drawn inspiration because the rima glottidis is spasmodically narrowed, so that the air enters the lower air passages very slowly. Feeble respiratory murmur is heard during the expiratory cough.

According to Guéneau de Mussy, there is dulness over the manubrium sterni on account of swelling of the tracheo-bronchial glands (?).

The patients suffer intolerable fear of suffocation during the attacks. But they often recover with surprising rapidity, and return to their occupation as if nothing had happened. Others suffer from exhaustion, dulness in the head, and slight vertigo for some time. There is frequent complaint of pain in the abdominal walls on account of strain in coughing.

In moderately severe cases, twenty to twenty-five attacks occur daily; in severe cases, as many as one hundred may occur in a day. They are always more frequent at night, because mucus is then more apt to accumulate in the air passages. According to Hanke, the attacks are more

frequent if the air is polluted with carbonic acid and ammonia, while exercise in the open air acts favorably.

The convulsive stage gradually passes into the decreasing stage. The paroxysms of coughing become less frequent, and lose their spasmodic character, so that the symptoms resemble an ordinary catarrh of the air passages. Exacerbations are apt to occur as the result of a cold, and the convulsive seizures again make their appearance.

The laryngoscopic changes readily pass into complications, although in my experience, as a rule, the laryngeal mucous membrane generally presents catarrhal changes. But as these changes are absent in many cases, I am inclined to attribute them to the violence of the coughing spells rather than to the specific influence of the pertussis virus.

As a rule, there is diffuse redness of the pharyngo-nasal cavity, larynx, trachea, and origin of the bronchi. The true vocal cords escape ; the anterior portion of the laryngeal cavity, as far as the true vocal cords, is sometimes not at all, always very little affected. The region between and beneath the arytænoid cartilages, and the anterior wall of the larynx and trachea below the true vocal cords, are inflamed with special intensity.

Croup and spasm of the glottis, which may prove fatal, are very rare complications on the part of the larynx.

Bronchitis is a frequent complication, and is present in the majority of cases. The situation becomes serious when this is complicated with pneumonia, as shown by accelerated, short, groaning respirations, and great rise of temperature. The alveoli sometimes rupture on account of the violent cough, and interstitial emphysema develops. If the pulmonary pleura ruptures, this may terminate in pneumothorax, or the air spreads along the peribronchial cellular tissue to the main bronchus, mediastinal connective tissue, and subcutaneous cellular tissue of the jugular fossa, where it appears as subcutaneous emphysema. The latter sometimes extends over a large part of the body. It is also possible that very extensive emphysema may produce danger of suffocation by compression of the air passages.

Struma, pleurisy, pericarditis, and endocarditis are rare complications.

There is often obstinate vomiting. The patients vomit after each spell of coughing, and, on the other hand, the ingestion of food produces a coughing spell. This may give rise to a serious condition of inanition.

Ulcers are sometimes found at the anterior edge of the frænum linguæ, more rarely on the inferior surface of the tongue. These are the result of mechanical injury by the teeth during the coughing fits.

The excessive strain of the abdominal muscles may result in hernia and prolapsus ani.

It is said that sugar is sometimes found in the urine. Steffen observed albuminuria during and immediately after violent coughing spells.

Characteristic cutaneous changes are often produced. On account of the pronounced venous stasis during the coughing fits, subcutaneous hemorrhages develop and occasionally attain considerable size.

Cutaneous œdema has been observed, but it is doubtful whether this is the result of marasmus or complicating nephritis. Pierson explains it as the result of acute dilatation and insufficiency of the right heart, *i. e.*, of stasis, and attributes to it a very unfavorable prognostic significance.

Hemorrhages of the mucous membranes are also observed frequently. Subconjunctival hemorrhages are very common; hemorrhages may also occur from the nose, air passages, and gastro-intestinal tract. Hemorrhages from the external auditory canal are sometimes observed as the result of rupture of the drum membrane.

Otitis media is present in rare cases; if bilateral, it may produce deaf-mutism in children under four years of age. In one case, Landesberg noticed optic neuritis and bloody infiltration of the lids; in another, exophthalmus of one eye from retrobulbar hemorrhage; in a third, hemorrhages into the retina and optic nerve, and in a fourth, dislocation of the lens.

In a few cases, there is marked change in the mental condition, amounting even to insanity. In rare instances, hemorrhages occur within the skull from venous stasis. Barrier reports a fatal case of subdural hemorrhage. Convulsions sometimes occur during a coughing spell from excessive venous stasis within the skull.

In some cases, grave sequelæ follow, and prove fatal at an early age. General marasmus is occasionally observed, and the children succumb after a longer or shorter period. They grow pale, emaciate, and finally die of exhaustion. Signs of scrofula sometimes develop. Chronic diseases of the respiratory tract (chronic hoarseness, bronchitis, or tubercular processes in the lungs) are not infrequently left over. Tuberculous, cheesy, bronchial glands may give rise to miliary tuberculosis, especially to tubercular meningitis. Pulmonary emphysema is sometimes produced by the acute distention of the lungs observed during the paroxysms, but as a general thing it soon disappears. Epilepsy and chorea are rare sequelæ. Certain complications (hernia, prolapsus ani, valvular lesions of the heart, auditory or visual disturbances) persist as sequelæ.

III. ANATOMICAL CHANGES.—Specific anatomical changes in pertussis are unknown.

The mucous membrane of the air passages is generally swollen and congested, and covered with abundant secretion. These changes may rapidly disappear in the dead body.

The tracheo-bronchial glands are often enlarged and congested. The congestion may also extend to the pneumogastric, which is embedded in the glands.

Pulmonary lesions are frequent and are often the cause of death. The upper lobes and median borders are pale and distended, the posterior and inferior portions contain congested and depressed non-aërated patches which are the result partly of atelectasis, partly of catarrhal pneumonia. Fibrinous pneumonia and fibrinous inflammation of the air passages are observed in rare cases.

The liver, spleen, and kidneys are congested and often slightly swollen. Similar changes are found in the mesenteric glands, the solitary glands and Peyer's patches of the intestines, and the follicles of the stomach. Ulcerations of the follicles of the intestines have also been mentioned.

Meningeal and parenchymatous hemorrhages may be present in the brain.

In our opinion, whooping-cough is the result of a general infection, *i. e.*, one

starting from the blood. This gives rise to increased irritability of the vaso-motor and cough centres in the medulla oblongata. The former causes increased secretion of the mucous membrane of the air passages, the latter increased excitability of the nerves of coughing, especially the superior laryngeal.

The theory that the disease is an ordinary central or peripheral neurosis is contradicted by its infectious character. This also disproves the opinion that it is an ordinary bronchitis with unusual irritability of the mucous membranes.

IV. Diagnosis.—During the convulsive stage the diagnosis is easy; and the laity properly lay great stress on the prolonged whistling inspirations. Obstinate coughing spells, attended with frequent vomiting and subconjunctival hemorrhages, or hemorrhages from other mucous membranes, must also arouse the suspicion of whooping-cough. This is also true of cough associated with ulceration of the frænum linguæ. In doubtful cases, it should be remembered that deglutition or depression of the tongue not infrequently produces an attack of coughing.

In the first and third stages of the disease, it is often difficult to make a differential diagnosis from ordinary bronchitis. The opportunity for infection is an important factor in diagnosis.

V. Prognosis.—This is usually good; the average mortality is about three per cent. The younger the patient the greater is the danger. The disease is also serious in anæmic, rachitic, and scrofulous children. As a general thing, more girls die than boys. The disease is more serious among the poorer classes than among the well-to-do, on account of insufficient nursing, overcrowding, and poor ventilation of the rooms. Sporadic cases are more favorable than epidemic ones. Grave complications on the part of the respiratory organs are more frequent in cold weather. The prognosis also depends on the daily number of coughing spells. According to Trousseau, the prognosis is serious if thirty to fifty attacks occur daily, and unfavorable if they exceed sixty. In pregnant women there is a possibility of premature delivery from the violence of the coughing movements, but the frequency of this event is often greatly overestimated.

VI. Treatment.—The spread of the disease can be prevented only by the strictest prophylaxis. The patients must be isolated, and the healthy children in the family are safe only when sent to another house. Half-way measures are useless. Isolation should be continued until the cough and catarrh have disappeared.

The children should have their own table service, their bedding and clothing should be cleaned separately, and they should be allowed to expectorate only in vessels containing carbolic acid (5%) or corrosive sublimate (1 : 1000). The physician should visit these patients last, in order that he may not convey the disease to other families.

Specific remedies against whooping-cough are unknown. The patient's room should be large and sunny, and well ventilated. Every two hours the air should be sprayed with a three-per-cent solution of carbolic acid. In winter, a vessel containing water and a half teaspoonful creasote should be placed on the stove. The air is kept at a uniform temperature of 15° R. If the weather is not windy, daily exercise should be taken in the open air. The diet consists of milk, eggs, soup, boiled meat, stewed fruit, and wine. Older children should be directed to repress the desire to cough as much as possible. In obstinate cases, removal to another locality sometimes produces favorable results with surprising rapidity, but the danger of producing infection in the new surroundings must be kept in mind.

Specially prominent symptoms must be treated according to general principles. If the number of coughing spells is very great, or their duration protracted, we may order narcotics, such as ℞ Aq. amygdal. amar., ʒ iij.; morphin. hydrochloric., gr. ss. M. D. S. 5 to 10 drops every three hours.

If there are numerous sonorous and sibilant râles, we may give expectorants, such as ℞ Sol. Apomorphin. hydrochlorat. gr. iss. : ℥ iiiss.; acid. hydrochloric., gtt. v.; syr. simp., ʒ v. M. D. S. ʒ i.-ij. every two hours; if there are numerous moist râles, we may order ipecac.

A combination of different dry and moist râles may be treated with ipecac and iodide of potassium, etc.

We may mention the following other remedies which have been employed in this disease. *a.* Nervines: valerian, musk, asafœtida, camphor, nitrate of silver, bismuth, arsenic, zinc and copper preparations, etc. *b.* Narcotics: chloral hydrate, chloroform, ether, croton chloral, veratrine, hyoscyamus, nux vomica, conium, ergotin, pulsatilla, cocaine, etc. *c.* Expectorants of all kinds. *d.* Antiparasitics: carbolic acid, salicylic acid, creasote, benzin, petroleum, quinine, breathing the air of gas houses. *e.* Balsamics: turpentine, ol. petræ italicum. *f.* Emetics, in repeated doses. *g.* Tonics: iron and quinine. *h.* Astringents: tannic acid, acetate of lead. *i.* Purgatives. *k.* Absorbents: potassium iodide and tincture iodine, applied to the manubrium (to disperse enlarged glands). *l.* Constant current to medulla and pneumogastric. *m.* Inspiration of compressed air. *o.* Derivatives to the chest. *p.* Specifics: cantharides tincture, propylamin, pilocarpine, powdered vaccine pustules internally, etc.

2. *Influenza.*

I. ETIOLOGY.—Influenza almost always occurs epidemically or pandemically, more rarely sporadically. The disease has sometimes extended over almost the entire globe, at other times only certain countries were attacked, or only certain cities or localities, such as crowded barracks, prisons, etc.

The majority of epidemics occur in winter, but very little influence is exerted by meteorological conditions. The epidemics often appear unexpectedly over large areas, and disappear in a few weeks with equal rapidity. They generally last four to six weeks, sometimes much less, but occasionally even eight to ten months. The disease sometimes spreads from one locality to a distant one, leaving the intervening district unaffected, or attacking the latter at a later date, or it appears at the same time in separate localities. Epidemics have been observed upon vessels at sea.

The disease occurs most frequently in middle and advanced life, and is also said to attack with special frequency those who live a good deal in the open air. Several attacks are experienced not infrequently by the same individual. A number of cases have been reported, in which individuals travelling to localities in which influenza was epidemic escaped the disease.

Whether the disease is conveyed from one individual to another is still an unsettled question.

Epidemics of other infectious diseases (measles, whooping-cough, varicella, small-pox, malaria) may also be present at the same time, but it has also been noticed repeatedly that, during influenza epidemics, the other diseases mentioned become less frequent or disappear entirely, sometimes reappearing towards the end of the influenza epidemic.

The nature of the virus is unknown. Letzerich described influenza-micrococci

in the blood (?). Seifert also described cocci in the nasal secretion and sputum, but they were absent in the blood and tears, and could not be conveyed to animals.

II. Symptoms.—The symptoms generally begin suddenly, more rarely they are preceded for a few hours or days by prodromata (malaise, pains in the limbs, somnolence or insomnia, gastro-intestinal disturbances, headache, etc.).

It is often maintained that the disease has no stage of incubation, but this is an open question.

The manifest symptoms are catarrh of various mucous membranes, fever, severe impairment of the cerebral functions, and striking weakness. All the mucous membranes may be affected in like degree, or the inflammation of one or the other may be specially prominent.

As a rule, the scene opens with a chill or repeated chilly sensations. The temperature rises, and the rapidity of the pulse is increased. The patients often complain of severe pain in the forehead near the glabella, more rarely of occipital pain. This may be associated with confusion of mind, delirium, convulsions, cramps in the calves, subsultus tendinum, tremor of the limbs, and a striking degree of prostration.

Hyperæmia of the conjunctivæ and epiphora set in, with photophobia. Then follow hoarseness, soreness in the trachea and beneath the sternum, cough (sometimes spasmodic)—in short, signs of catarrh which begins above and gradually descends. Finally, bronchitis also develops. Attacks of nervous dyspnœa occur not infrequently. Coated tongue, a bad taste in the mouth, anorexia, vomiting, constipation, or more rarely diarrhœa indicate gastro-intestinal catarrh. If meteorism is also present, a suspicion of typhoid fever may be aroused.

The disease sometimes terminates in two to six days, or it may last two weeks. The sudden occurrence of diaphoresis may indicate a crisis; in other cases, the patient sweats during the entire course of the disease. Unusual prostration is generally left over for a considerable period.

The following complications have been observed: broncho-pneumonia, more rarely fibrinous pneumonia, pleurisy, even pericarditis, exceptionally croup. Erythema, roseola, urticaria, miliaria, petechiæ, and aphthæ of the mucous membrane have also been described. Ptyalism and parotitis have been observed in exceptional cases.

Phthisis, secondary to chronic broncho-pneumonia, may develop as a sequel.

III. Anatomical Changes.—The lesions are similar to those of the corresponding disturbances when independent of influenza.

IV. Diagnosis and Prognosis.—In view of the sudden and epidemic occurrence of the disease, the diagnosis is easy. The prognosis is good in middle life, but is serious in adults and when complications develop. It often aggravates previously existing diseases. Pregnant women are exposed to the danger of premature delivery.

V. Treatment is purely symptomatic. Many recommend quinine almost as a specific. Narcotics may be given to relieve the cough, expectorants to facilitate expectoration, emetics if gastric symptoms are prominent, etc.

3. *Hay Fever. Summer Catarrh.*

I. Etiology.—This disease occurs as catarrh of the air passages, conjunctiva and lachrymal canal, and as asthma (so-called hay asthma).

It is most frequent between the ages of fifteen and thirty years, rarer in childhood; it never develops after the age of forty years. Men are attacked about twice as often as women.

The educated classes are almost always attacked, the laboring classes rarely. A distinct predisposition is necessary to the development of the disease; this may be congenital or acquired. The patients generally come of neuropathic families, or have been rendered nervous by various causes. They present increased irritability of the vaso-motor system, in consequence of which slight irritation of the mucous membranes produces hyperæmia and inflammatory changes. According to Hack and Roe, abnormal swelling of the mucous membrane over the inferior turbinated bone, also over the middle turbinated bone and the septum, often give rise to hay fever.

In many persons, the symptoms occur periodically at definite times, usually between May and September. As a rule, the cases are more frequent between May and July.

It has long been recognized that the outbreak of the disease coincides with the flowering of grasses, and individuals are often attacked immediately after approaching a mowed lawn, a field of grain, etc. It is now held that the disease is produced by the pollen of flowering grasses. As the pollen is diffused far and wide, it is not astonishing that individuals should be attacked, although not exposed apparently to the exciting cause.

This theory is confirmed by the fact that the symptoms coincide in point of time with the first and second crops of hay; that the secretion of the inflamed mucous membranes has been found to contain grains of pollen, and that the application of the latter to the nasal mucous membrane of predisposed individuals will produce the disease. The development of the disease is favored by drought and winds, and prevented by steady rains. People living in the country generally escape the disease, but this is probably owing to the absence of the predisposition.

I. Symptoms.—The symptoms not infrequently begin immediately after a walk across a meadow, *i. e.*, the cause is immediately followed by its effect. Prodromata (malaise, anorexia, slight fever, etc.) are observed occasionally for several hours or days.

In the catarrhal form of hay fever, the first symptoms are those of acute coryza: burning and pricking in the nose, frequent sneezing, increased nasal secretion; olfactory hyperæsthesia is sometimes noticeable. Then follow symptoms of conjunctival catarrh, "foreign body" feeling in the eyes, increased lachrymal secretion, photophobia, injection of the conjunctiva, œdema of the lids. Then scratching and dryness of the throat, and slight difficulty in deglutition. These symptoms may or may not be followed by catarrh of the larynx, trachea, and bronchi. There is sometimes slight fever, often mental depression, and severe pain in the forehead or occiput. The patients sometimes have an annoying sensation of coldness in the nose, especially its tip, which may be cold to the feel. In one of my colleagues, the integument of the nose always became red and contained dilated vessels. Extensive erythema and urticaria have also been observed. The symptoms often last three to eight weeks, rarely only a few hours or days. As a rule, relapses occur.

Pollen of graminæ has been found in the nasal and lachrymal secretions, either intact or swollen and ruptured. The contents are described as very fine, moving granules, often arranged in chains.

Hay asthma corresponds in its clinical history to a full-developed asth-

matic attack. In the bronchial secretion, Schmidt found Leyden's asthma crystals, in addition to grains of pollen. Transitions may occur between the two varieties of hay fever.

III. Anatomical Changes, Diagnosis, Prognosis.—The anatomical changes are visible on inspection in the living subject. They consist of marked congestion, swelling, and hypersecretion of the affected mucous membranes. The disease rarely terminates fatally, and only one fatal case (in an old man) has come to my knowledge.

The diagnosis is easy, in view of the causation and regular recurrence of the symptoms. The prognosis is unfavorable so far as regards permanent recovery.

IV. Treatment.—The treatment should be chiefly prophylactic. If the individual suffers from excessive erectility of the nasal mucous membrane, the galvano-cautery should be employed. Predisposed individuals should live during the summer at the sea-shore, or on high mountains. Existing nervousness should be combated by a rational mode of life, cold rubbings, baths and douches; when exposed to infection, the nose should be douched every two hours with salt water, or a solution of carbolic acid or quinine, in order to remove the inspired pollen as rapidly as possible.

Irrigation of the nasal cavity with a solution of quinine (1 : 740), carbolic or salicylic acid, and other disinfectants, may be performed when the disease is fully developed.

In one of my cases, good effects were obtained from the following snuff powder: ℞ Hydrarg. chlorid. mite, alumin., āā gr. xlv.; morphin. hydrochlorat., gr. ivss. M. D. S. A portion as large as a pea to be snuffed up t. i. d.

Nervines (bromide of potassium, arsenic, strychnine, and camphor) and central galvanization have been employed in order to relieve the general nervousness. Narcotics may be administered internally and locally if the symptoms of irritation are violent.

PART V.

INFECTIOUS DISEASES IN WHICH THE DIGESTIVE APPARATUS IS CHIEFLY INVOLVED.

1. *Epidemic Parotitis. Mumps.*

I. Etiology.—Epidemic parotitis occurs particularly in the winter and autumn, and its spread seems to be furthered by unfavorable weather. It is sometimes a forerunner of epidemics of measles, whooping-cough, or diphtheria, or it follows these diseases. It is sometimes prevalent at the same time as epidemics of scarlatina.

Parotitis is said to be endemic in certain places (coasts of Holland, France, and England, certain parts of the United States,)

The male sex is affected more frequently than the female sex. Certain epidemics attack children almost exclusively, in others adults are chiefly affected. The disease does not occur in infancy or old age. It is most frequent between the second and twentieth years.

It develops very often in crowded institutions. Among 131 pupils in the Cadet Academy at Ploeñ 118 (90%) were attacked.

The contagious character of the disease has been demonstrated in a number of cases. For example, an apparently healthy individual travels from a locality in which parotitis is epidemic to another district. Here he is attacked by mumps at the end of a few days, and soon infects those living in the same house, and then those in neighboring houses.

Epidemic parotitis is probably a general infectious disease, characterized in the main by a local affection of the parotid gland. This opinion is supported by its epidemic character and contagiousness, and the fact that a single attack generally confers immunity in subsequent epidemics. The general symptoms are often very slight, but cases occur, on the other hand, in which internal organs are seriously affected, while clouded consciousness, high fever, and great prostration produce the impression of a serious infectious disease.

It is plausible to assume that the virus passes from the buccal cavity through the excretory duct of the parotid, and that the general symptoms depend upon the unobstructed passage of the virus into the general circulation. Soltmann believes that the narrowness of Steno's duct in infants, together with the slight development of the salivary glands, confers immunity upon infancy.

Pasteur found in the blood rod-shaped bacteria 1 μ broad and 2 μ long, but attempts at inoculation in animals proved fruitless.

II. Anatomical Changes.—According to Virchow, the lobules of the parotid gland are hyperæmic, the excretory duct is filled with mucopus, and the periglandular connective tissue is in a condition of inflammatory œdema and infiltrated with round cells.

III. Symptoms.—The duration of the stage of incubation is variously estimated at nine to twenty-one days. The characteristic symptoms are sometimes preceded by prodromata for one to three days. The patients complain of fever, prostration, and anorexia. In children the rise of temperature (sometimes to 39.5° C.) may cause eclamptic convulsions.

The first specific changes consist of pressure and tension in the region of the parotid gland. Violent pains sometimes occur, and may radiate into the auricular region, and between the shoulder blades. The movements of the jaw are interfered with. The parotid region soon becomes swollen, the swelling extending in all directions beyond the gland, upwards to the eyelids, downwards to the clavicle, posteriorly to the spine, and anteriorly to the chin. As a rule, the swelling is pale, the overlying integument smooth, glossy, sometimes œdematous. In rare cases, it presents inflammatory redness. It is hot to the feel, of a doughy hardness in consistence, and more or less tender on pressure.

The changes begin on one side (generally the left), but later extend to the other side.

The swelling abolishes the ability to perform mimetic movements, and gives to the patients a peculiarly stupid appearance. If the disease is unilateral, the head is generally held immovable towards the affected side, and attempts at rotation produce extremely violent pains. In bilateral mumps, the head is held peculiarly stiff, and is somewhat bent over forwards or backwards. The movements of the lower jaw are almost entirely abolished, and sometimes only a narrow fissure is left

between the rows of teeth. Hence the patients are restricted almost exclusively to fluid food. Articulation is rendered indistinct, and fœtor ex ore is often produced on account of the inability to keep the mouth clean.

The latter symptom is intensified when stomatitis and ptyalism develop. The secretion of saliva is often diminished at the outset, and is only increased in the later course of the disease. Tonsillitis, pharyngitis, and follicular inflammations are also observed in some cases.

The swelling is the result, not alone of changes in the parotid, but also of inflammatory œdema in the surrounding cellular tissue. The submaxillary and sublingual glands are also inflamed in some cases, and it is said that these glands may alone be swollen during on epidemic of mumps. The adjacent lymphatic glands generally take part in the swelling.

After the swelling has formed, the fever generally falls and remains below 39° C. Some cases run an apyrexial course.

An uncomplicated case lasts about two weeks. The swelling gradually diminishes, the movements of the jaws become freer, and finally recovery is complete. The skin often desquamates over the site of the tumor.

The most important complications are affections of the sexual apparatus. Orchitis develops not infrequently in male adults. Granier found that among 495 cases in an epidemic among soldiers, orchitis developed 115 times. Children and old men are not attacked in this way.

The orchitis is generally unilateral, often upon the side on which the parotid is mainly or exclusively affected. In bilateral mumps, the right testicle is more frequently inflamed. Bilateral orchitis is rare, but sometimes one testicle is attacked after the other. The first symptoms consist of pains in the loins, groins, and along the seminal cord. The temperature of the body is generally increased, and vomiting occurs. After a time, the testicle is found to be enlarged and tender on pressure; the scrotum is generally red and œdematous. The testicle itself, not the epididymis, is inflamed.

The frequent complication with orchitis depends on the character of the epidemic. Blondeau reported a case in which, although mumps developed while the patient was suffering from gonorrhœa, orchitis did not occur. Nor does orchitis depend upon the severity of the inflammatory changes in the parotid. Rizet described an epidemic in which the mild cases gave rise to orchitis.

In a number of cases, the parotitis subsides on the appearance of orchitis, or both lesions present repeated variations in severity. In some cases of parotitis, a gonorrhœa-like discharge takes place from the urethra.

Pains and even enlargement of the ovaries, swelling, and hæmatoma of the vaginal mucous membrane and labia, and enlargement of the breasts have been observed in female patients.

Suppuration of the inflamed parotid gland is an unpleasant complication. The pus may perforate externally through several fistulæ or into the mouth, pharynx, or external auditory canal; or it destroys the facial nerve and produces permanent facial paralysis; or erodes the large vessels in the neck and causes fatal hemorrhage; or ruptures into the air passages, pleural or pericardial cavities, etc.; or finally it produces pyæmia or septicæmia.

Among other complications we may mention: *a*. Cerebral congestion (from pressure of the parotid on the jugular vein) and meningitis. *b*.

Delirium and maniacal attacks, which always have an unfavorable prognostic significance. *c.* Visual disturbances (amblyopia and color blindness) have been observed a number of times by Falry, who attributes them to congestion of the papilla. Conjunctivitis, epiphora, and photophobia may also occur. *d.* Laryngeal stenosis, resulting from compression of the larynx, was observed in some epidemics. *e.* Acute albuminuria, uræmia, and death were described by Colin. Hæmaturia and persistent albuminuria have been observed. *f.* Gastro-intestinal disturbances are very frequent; the bowels are generally constipated. Obstinate vomiting is observed in some cases. *g.* Isham claims to have observed endocarditis (?).

Sequelæ are sometimes left over. The inflammatory swelling may not be entirely absorbed, certain portions persisting for many months or even for life. This is observed with special frequency in scrofulous children. Suppuration of the parotid is also apt to follow in them, and this may be followed by the formation of a fistula, burrowing of pus, pyæmia, or erosion of large vessels. In one case I noticed severe ptyalism which lasted three months and was cured by atropine. Burton observed absence of salivary secretion and relieved the condition by the application of the constant current. Facial paralysis may be produced by pressure on the facial nerve. Atrophy of the testicle sometimes occurs in males who have suffered from orchitis. In Granier's 115 cases of orchitis, 51 suffered from atrophy of the testicle. Jaloux described a case in which the healthy testicle underwent hypertrophy. Deafness has been observed, preceded by noises in the ears and vertigo. It seems to depend on primary or secondary affection of the labyrinth or on secondary catarrh of the middle ear.

IV. Diagnosis.—The diagnosis is easily made from the presence of a swelling between the mastoid process, external auditory canal, and zygomatic process, and from the epidemic character of the affection.

V. Prognosis.—The prognosis is almost always favorable. It is only in rare cases that life is endangered or that serious sequelæ persist.

VI. Treatment.—Oily inunctions and the application of cotton batting are almost always sufficient in the way of treatment. The bowels should be opened daily, and the diet consist chiefly of fluid food. Lestor recently recommended the administration of jaborandi. If orchitis develops, the patient must remain in bed and keep the testicle elevated. If suppuration of the parotid occurs, it should be poulticed and opened as soon as possible. Indurations are treated by applications of iodine tincture or ointment, iodoform ointment, and iodide of potassium or iron given internally.

2. *Ephemeral Infectious Fever.*

(*Herpetic Fever.*)

I. Symptoms.—Every physician probably meets cases in which a general febrile condition suddenly develops, often without any known cause, and disappears in a short time. The scene opens not infrequently with a single violent chill, or the patients complain of repeated chilly sensations, great prostration, apathy, and a feeling as if a severe illness is impending. In children, convulsions may develop. The bodily temperature soon rises to 39°, 40° or more. The pulse and respirations are accelerated, thirst increased, diuresis diminished. The tongue generally

presents a thick, gray, yellow, or brownish coating, and there is often a disagreeable fœtor ex ore. Eructations and vomiting are not uncommon. There is sometimes tenderness over the stomach, and meteorism.

Herpes often develops very rapidly on the lips, rarely on the ala nasi, cheek, lobe of the ear, or other parts of the body. In a few cases I found a few patches of roseola on the abdomen. Even slight impairment of consciousness and delirium may set in.

The disease often terminates in the following night. Profuse diaphoresis occurs, and the temperature becomes normal or even sub-normal, an abundant sediment appears in the urine, and the existing feebleness rapidly disappears. In rarer cases the disease lasts several days, very rarely more than a week.

II. Etiology.—Many individuals exhibit a predisposition to such attacks, and may suffer from a large number in the course of their lives. They are most frequent in middle life. They are generally attributed by the patients to a cold, but, in the majority of cases, without justification. Others attribute the disease to excesses in eating ordrinking, mental or physical strain, or the action of excessive heat. Several individuals are sometimes attacked at the same time, or in succession, so that, in our opinion, we are justified in regarding the disease as an infectious fever of ephemeral duration. Plessing has recently reported a case in which several patients were attacked in the same ward of a hospital.

III. Diagnosis, Prognosis, Treatment.—The diagnosis is easy. Apart from the febrile and gastro-enteric symptoms, affections of other organs are absent, and the rapid and almost always favorable course differentiates the disease from typhoid fever, central pneumonia, etc.

The prognosis is almost always favorable.

The treatment is purely symptomatic.

3. *Typhoid Fever.*

I. Etiology.—Sporadic cases of typhoid fever are found almost constantly in large cities. In certain countries and cities the disease is especially frequent. While it is very common in England, typhus fever predominates in Scotland and Ireland. Among German cities, Munich was formerly notorious as the habitat of typhoid fever.

At times the disease appears in epidemics. These sometimes appear in places which were previously free from the disease, resulting from an imported case, from the ingestion of infected food, or apparently spontaneously.

Epidemics often occur during wars, their spread being favored by deprivation, colds, etc.

The majority of recent writers believe that the virus consists of certain low organisms (vide Fig. 30). Gaffky found typhoid bacilli in the intestinal ulcers, mesenteric glands, spleen, and blood-vessels of the liver and kidneys. They were situated in groups, and were so much more numerous the more recent the disease. Pfeiffer recently succeeded in cultivating them from the fæces. They have never been found in the blood.

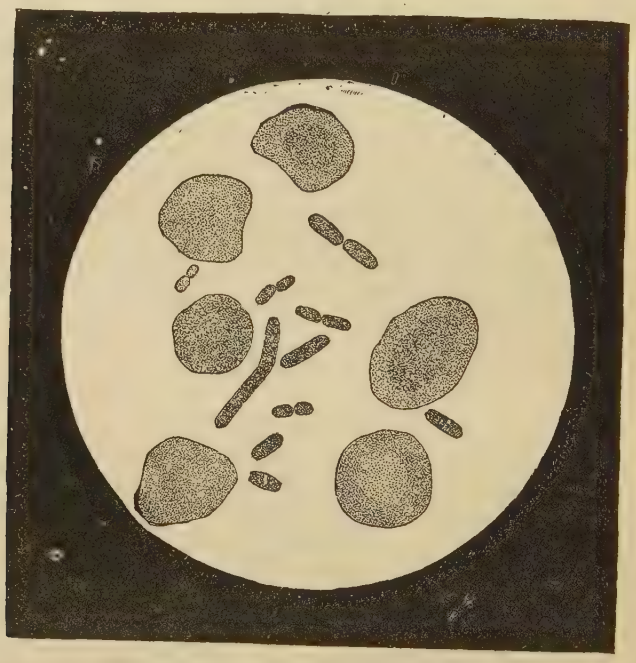
The length of typhoid bacilli is about one-third the diameter of a red blood-globule, their width about one-third their own length. The ends are rounded. They are readily stained with aniline, for example, methyl blue, gentian violet, Bismarck brown, and fuchsin. They occasionally contain spores, which do not stain with aniline colors. The spores are as broad as the bacilli, and are always situated near one extremity of the bacillus. Each bacillus contains only one

well-developed spore. If two bacilli are adherent, the spores are found only at the adjacent extremities.

Gaffky cultivated the bacilli in meat extract, peptone-gelatin, and slices of potato, but inoculations in animals did not produce positive results. Hence the results of his investigations may only be regarded as probable, not as certain.

Clinical experience indicates that the virus is contained in the fæces, so that the danger of infection arises only from contact with the intestinal contents. The virus is not diffused in the exhalations of the patient, so that it is not conveyed to those who come in contact with him. Hence the patients may safely be kept in the general wards of a hospital, if care be taken to disinfect the fæces, bed-pan, and soiled articles of clothing.

FIG. 30.



Typhoid bacilli from the juice of the mesenteric glands. After Eberth. Enlarged 1,980 times. Oil immersion.

The virus may enter the intestinal tract through the anus or mouth. If the infection occurs through the agency of water-closets or bed-pans which have been soiled by typhoid stools, infection takes place in the former method; but sometimes the virus is introduced in the ingesta, or it is diffused from the fæces into the air, enters the pharynx, and then is swallowed. Some assume the possibility of infection through the respiratory organs. Certain cases in which the disease begins with severe inflammation of the lungs would seem to indicate that the virus may first produce its injurious effects at its site of entry (the lungs) before exercising its full deleterious influence on the intestinal canal.

Drinking-water undoubtedly plays a prominent part in infection. Epidemics often affect only those houses or streets whose inhabitants

obtain their drinking-water from a certain well. In a number of such cases, it has been found that the water supply communicated with water-closets, dung heaps, ditches, etc., which contained infected fæces.

A number of cases have been observed, particularly in England, in which the disease was confined to families who received their supply of milk from a certain milkman. In the majority of these cases the milk had been diluted with infected water.

In the canton of Zurich a number of epidemics have been observed, which have been attributed to the ingestion of spoiled meat. These generally occurred at festivals, the spoiled meat being used in the preparation of special popular delicacies. In the Kloten epidemic (1878), among 700 singers 500 were attacked by typhoid fever. These epidemics have given rise to much controversy, especially upon the following points: *a*. Was the disease really typhoid fever? *b*. If so, was it produced by meat which was simply spoiled? Or *c*. By infection with the meat of typhoid animals? My colleagues in Zurich are agreed as regards the typhoid nature of the epidemics, and the statements of Wyss will hardly admit of any doubt. Huguenin states that the Kloten epidemic was produced by eating the flesh of a calf which suffered from typhoid fever, but fails to prove the truth of this assertion. Positive evidence that typhoid fever occurs in animals has not been furnished hitherto. But to assume that the disease was produced by the mere ingestion of spoiled meat contradicts the theory of the specific nature of the typhoid virus. Moreover, we know that the ingestion of spoiled meat produces symptoms (hemorrhagic gastro-enteritis) which differ from those of typhoid fever. It must be remembered that the participants in the festivals in question not alone ate, but also drank, and it is not at all improbable that the infection occurred through the medium of the drinking-water.

In some cases infection is attributable to certain occupations, for example, that of washerwomen, who wash clothing which has been soiled by the excrement of typhoid fever patients, or scavengers who clean water-closets, etc. It must be kept in mind that the virus may retain its potency for many years, and thus it often becomes difficult to ascertain whether typhoid stools have ever been emptied into the closets.

The outbreak of an epidemic is sometimes caused by an imported case, but in other cases the mode of infection cannot be discovered. In our opinion, there are mild forms of the disease in which the patients walk about. The fæces of such individuals may form the starting-point for severe cases. Indeed, cases have been reported in which fully developed lesions of typhoid fever were found upon autopsies on individuals who had been killed by accidents while apparently healthy.

The majority of cases occur from August to November, the fewest from February to April. Exceptions to this rule are sometimes noticed. In Munich, for example, the largest number of cases occur in the month of February.

As a general thing, cases are more numerous after a hot summer, while the spread of the disease is antagonized by a very cold winter.

According to Buhl and Pettenkofer, typhoid fever increases the lower the level of the subsoil water. They assume that the typhoid virus then proliferates more abundantly in the upper, dry strata of the soil, and are thus conveyed to the lower strata of the atmosphere. This theory, however, is opposed by numerous experiences.

To explain the occurrence of typhoid cities, we must assume that, in the course of years, the subsoil becomes saturated with typhoid germs which are constantly proliferating. As a matter of course, the level of the subsoil water may be important, but only because the higher its level the more apt it will be to contaminate the wells.

Typhoid fever very often occurs in house-epidemics (barracks, prisons, etc.). In large cities, certain houses and streets are apt to be notorious as the habitat of the disease. Extensive epidemics are sometimes found to consist of a number of house-epidemics.

The disease is most frequent from the fifteenth to the thirtieth years (maximum from twenty to twenty-five years), although it also occurs in childhood and old age.

Charcelay claims to have observed it twice in the new-born. Hastelius reported the case of a pregnant woman suffering from typhoid fever, who had a premature delivery in the eighth month. In the still-born child were found enlargement of the spleen and lymphoid infiltration of the intestinal follicles and mesenteric glands. Typhoid fever is rare in the first year of life. During childhood, it is especially frequent from the age of five to ten years.

Sex exerts no special influence on the frequency of the disease, though, on the whole, more men are attacked than women.

Robust individuals are often attacked, and some physicians believe that weak ænæmic individuals, and those suffering from cancer, heart-disease, syphilis, or phthisis, possess almost complete immunity.

It was formerly believed that pregnancy and the puerperal condition confer immunity against typhoid fever, but this view is now discarded. Indeed Hecker believes that the susceptibility is increased in these conditions. According to Duguyot, premature delivery is produced in two-thirds of the cases which occur in pregnant women. The child is generally still-born, or dies soon after birth. As a rule, pregnancy does not affect the course of typhoid fever, and complications often remain absent, even after premature delivery. The latter may be the result of the high fever, the severity of the infection, or disturbances of respiration.

Pfeiffer believes that typhoid fever is unusually frequent in certain families, and assumes that this fact is owing to the diminished power of resistance of the intestinal follicles.

The disease is most frequent among the poor. A predisposition may also be created by certain occupations. For example, the physicians or nurses in a hospital escape, while the washerwomen employed to wash the clothing of typhoid fever patients fall victims to the disease.

Strangers are especially apt to be attacked when they enter an infected city or house, while the inhabitants gradually become acclimated.

As a rule, only a single attack is experienced, although exceptions have been observed, and in a few cases individuals have suffered even three attacks.

Typhoid fever is sometimes associated with, or followed by other infectious diseases.

The combination with measles, scarlatina, typhus fever, variola, acute articular rheumatism, and intermittent fever has been reported.

During an epidemic, the individual cases often resemble one another very closely in their symptoms and general course.

II. Anatomical Changes.—The specific lesions of typhoid fever affect the lymph follicles of the intestinal mucous membrane, the mesenteric glands, and the spleen.

The intestinal changes generally begin with catarrhal inflammation, not alone of the lymph follicles, but also of the mucous membrane, and the latter persists in greater or less degree during the entire course of

the disease. The catarrhal follicles at first project more strongly from the surface, and are generally surrounded by a zone of hyperæmic vessels. The solitary follicles not infrequently look like fine pearls, which, if punctured, discharge a clear fluid. It is evident that the swelling is the result of inflammatory œdema. But soon the cellular elements proliferate, the follicle becomes more opaque and cloudy, and no longer collapses when punctured. This constitutes the acme of the catarrhal stage (second half of the first week of the disease).

There is now a gradual transition into the stage of medullary infiltration. The hypertrophy and hyperplasia of the cellular elements constantly increase, so that the solitary follicles may attain the size of a pea or more, while Peyer's patches form large plaques, whose thickness may exceed five mm. The edges of the infiltration usually are steep, but occasionally it sprouts up like a mushroom, the resemblance being increased still further by a slight depression in the centre. In Peyer's patches, the proliferation of the follicular substance proper not infrequently greatly exceeds that of the interfollicular connective tissue, so that its surface presents a latticed appearance. In some places the infiltration is confined to certain parts of the patch, while adjacent parts maintain the normal appearance. If the morbid process is very extensive, adjacent follicles may coalesce. In this way tumor-like masses may form upon the mucous membrane, and may narrow the lumen of the canal to a certain extent. This is observed with relative frequency on the ileo-cæcal valve and lower part of the ileum.

The medullary infiltration often extends beyond the follicles to the adjacent mucous membrane, in places to the muscular coat, and even to the serous layer. In this way small medullary nodules may form beneath the peritoneal covering of the intestines. Even if these changes are not produced, the serous membrane over the typhoid changes is often congested.

At first the infiltrated places are congested and succulent, but later the congestion disappears, the parts assume a grayish-red, then a white color, and become firm, almost crumbly.

Under specially favorable circumstances, the infiltration may undergo absorption. The cellular elements undergo fatty degeneration, and the fatty detritus enters the lymphatic channels. This is shown macroscopically by the yellow color of the parts. In Peyer's patches, it is found not infrequently that the cellular elements in the follicular substance undergo fatty degeneration and absorption earlier than in the interfollicular connective tissue, so that the patch presents a furrowed appearance, the follicles being depressed, and the interfollicular bands of connective tissue forming prominent ridges. If the medullary infiltration has been attended with extravasations of blood, the coloring matter of the blood is gradually converted into blackish pigment detritus. The Peyer's patches may then present a black speckled appearance. These changes generally persist for many years. Extravasations may also form in the hyperæmic zones which surround the follicles, and may be followed by pigmentation.

As a rule, however, the stage of medullary infiltration passes into that of necrosis, or at least this is true of the majority of the infiltrated lymph follicles. As a general thing, the necrosis takes place about the middle of the second week of the disease. This is evidently owing to the fact that the cellular elements increase in such numbers as to compress the blood-vessels, and thus deprive the cells of nutriment. A thin layer

of necrosis first forms on the surface of the follicle, and assumes a yellowish or brownish color from imbibition of bile pigment. But the necrosis may extend so deeply as to lead to perforation of the serous layer. The mucous membrane adjacent to the lymph follicles occasionally undergoes necrosis, and large gangrenous shreds sometimes project into the lumen of the intestines.

About the middle of the third week the stage, of ulceration begins. The necrotic masses are exfoliated, and leave a loss of substance, at the bottom of which the muscular coat is distinctly visible. Ulcers of the solitary follicles are usually round, those of Peyer's patches are oval in shape. Unlike tubercular ulcers, their long diameter is parallel to that of the intestines, and annular ulcerations are almost always absent. Exfoliation generally occurs in very minute particles. More rarely large pieces, or the entire necrotic patch are exfoliated, and may then be found in the stools during life. Ulceration and exfoliation may be attended with danger, particularly with hemorrhage from large vessels. As a general thing, however, this is prevented by thrombosis of the vessels.

The stage of recovery begins about the middle of the fourth week. Granulations form upon the base of the ulcer (sometimes so actively as to almost constitute suppuration) and gradual cicatrization ensues. This rarely results in intestinal stenosis, although the adjacent mucous membrane takes part in the process, as is evident from the slightly radiating character of the cicatrix. For years the cicatrix forms an attenuated patch which appears with special distinctness when the intestine is held up to the light. Its centre or border is not infrequently pigmented. The cicatricial tissue is sometimes lined merely with a layer of epithelium; in other cases, a new formation of villi with blood-vessels takes place; the villi are generally scanty, and of unequal height and width.

The microscopical changes in the follicles affect all their constituents. The vessels are dilated, their walls in a condition of vitreous swelling. In places they are plugged with white blood-globules. The parenchyma cells of the follicles present active fission and proliferation; giant cells are found containing ten to fifteen nuclei (so-called typhoid cells of Rindfleisch). In the connective-tissue stroma are found swelling of the tissue, proliferation of the branching cells, and infiltration with round cells. Typhoid bacilli probably form an integral part of the changes. Heschl observed that the changes extended far beyond the lymph follicles. In the capillaries of the intestinal walls he found swelling and proliferation of the nuclei, so that they projected in places into the lumen. Similar changes were found in the muscle nuclei of the longitudinal muscular layer of the intestines, where they had produced, in places, nests of round cells.

The typhoid changes in the follicles begin and are most marked in the lower end of the ileum and upon the valve. They are no longer found in the duodenum, although some authors claim to have found typhoid changes even upon the mucous membrane of the stomach. The vermiform appendix is sometimes attacked with special severity, and in one of my cases of perforation-peritonitis a typhoid ulcer was found only at the tip of the appendix. The large intestine may escape entirely; when attacked, the solitary follicles are affected, since it does not contain any Peyer's patches. At times, the disease may extend into the rectum, and large losses of substance may result from the coalescence of adjacent ulcers. The large intestine is sometimes the chief site of typhoid fever. The changes in the follicles are often found in various stages of development.

The affection of the intestinal lymph follicles is attended with changes

in the mesenteric glands. These appear earliest and are most marked near the lower part of the ileum. Some of the mesenteric glands may attain the size of a pigeon's egg or even a hen's egg. On section, they are found to be very red, more markedly in the cortical substance. Extravasations of blood may occur in places. The cut surface is moist and juicy, the consistence soft. Later, the congestion diminishes, the succulence partly disappears. and a condition of medullary infiltration develops, as in the lymph follicles of the intestines. In teased preparations, cells containing blood-corpuscles have been found not infrequently. Bacilli have also been found here. As the changes upon the mucous membrane subside, the swelling of the mesenteric glands diminishes; they undergo fatty degeneration and absorption. In some cases, spots of necrotic softening form in the glands, and may give use to perforation with subsequent peritonitis. Or caseation and calcification may develop; this may terminate finally in infection with tubercle bacilli and in general miliary tuberculosis.

The spleen begins to enlarge about the middle of the first week, and reaches its greatest dimensions about the end of the second week. This enlargement may not occur in old people or in cases in which the splenic capsule is thickened or firmly adherent to adjacent organs. At the height of the changes the capsule is tense; when diminution in size occurs, it is often wrinkled. The consistence of the organ is soft, occasionally almost diffluent. It is distended with blood, of a dark cherry-red color, and the follicles sometimes appear upon the cut surface as small gray infiltrations. In advanced cases, there is often an unusual amount of pigment in the spleen, the result of unusual destruction of red blood-globules within the organ. In addition to evidences of proliferation and fission of the splenic cells, the microscope also shows an unusual number of these cells which are filled with more or less changed red blood-globules or with their detritus. Typhoid bacilli have also been found in the spleen. Wedge-shaped and simple hemorrhagic infarctions, and even abscess of the spleen are not very rare complications.

It would appear as if almost the entire lymphatic gland system may be affected in severe cases. Thus, medullary infiltration has been observed, not alone in the retroperitoneal, but also in the tracheo-bronchial and even the peripheral glands (cervical and inguinal). Similar changes have been observed in the tonsils, the follicles of the tongue, and the thyroid gland. The medulla of the bones is often of a red color, its parenchyma cells increased in number; it contains an unusual number of nucleated blood-globules and cells which contain blood-corpuscles.

The specific lesions mentioned above are always associated with changes in other organs, partly as the result of infection, partly owing to the increased bodily temperature.

If the patients have died at the height of the disease, rigor mortis is apt to develop very quickly and to be very marked. Livores mortis are usually very abundant upon the back and dependent portions of the body.

The panniculus adiposus is often well retained, even if the disease has lasted two or three weeks. The muscles are generally dry, and have a deep red color. Here and there they contain pale-gray or yellowish patches, which are most frequent in the recti abdominis and adductors of the thigh, but are also found in other muscles (even the heart, tongue, and diaphragm). In such places the microscope shows that the contents of

the muscular fibres have a dull shining, vitreous, coagulated appearance, and are converted into fibrillated clumps (Zenker's degeneration). In other places the muscular fibres contain very fine granules, which partly disappear on the addition of acetic acid (albuminoid), but partly persist and turn black on the addition of hyperosmic acid (fat granules). Proliferation of the muscle nuclei is observed not infrequently.

The heart muscle is often flaccid, brittle, and pale. On section, it is found to contain numerous pale-gray and light-yellow spots, which show under the microscope cloudy swelling of the muscular fibres, fatty and wax-like degeneration. The fibres often contain an unusual amount of brownish pigment. Nuclear proliferations within the fibres are also observed.

The larynx may contain ulcers, which are situated most frequently on the edges of the epiglottis or the posterior portion of the true vocal cords. These are, perhaps, the result of typhoid infiltration, at all events they often contain typhoid bacilli. They sometimes extend to the cartilages, producing necrosis and exfoliation, or they may give rise to fatal œdema of the glottis. Bronchitis is observed in almost every case.

The salivary glands are often enlarged, especially in the first stages of the disease. The microscope shows proliferation and cloudy swelling of the cells within the acini. The signs of pharyngitis, which are usually present during life, disappear in great part after death. Catarrhal gastritis is found not infrequently. The pancreas presents similar changes to the salivary glands.

The liver is often enlarged, its cells in a condition of cloudy swelling or fatty degeneration. Wagner observed lymphomata in the liver and kidneys. The gall-bladder is often flabby, and contains thin bile which is poor in coloring matter.

The kidneys are often slightly swollen, not infrequently pale and flabby. The microscope reveals cloudy swelling and fatty degeneration of the tubular epithelium. There is sometimes slight catarrh of the mucous membrane of the urinary passages.

The brain may present meningeal hemorrhages and œdema of the pia mater. Meynert found distention of the cortical capillaries, granular degeneration of the ganglion cells, increase of their nuclei, and fibrillation of the protoplasm. Popoff observed infiltration of the ganglion cells with round cells, and an accumulation of round cells in the perigangliouic lymph spaces, adventitious lymph spaces of the blood-vessels, and along the nerve fibres. This author also observed pigment infiltration of the ganglion cells. In places the pigment granules are free, and are visible to the naked eye as yellow or brownish patches.

Fatty degeneration of the blood-vessels is found in the brain and other organs.

III. Symptoms.—The duration of the stage of incubation probably varies from fourteen to twenty-one days, although, as in the case of other infectious diseases, it is sometimes longer, sometimes shorter than this period.

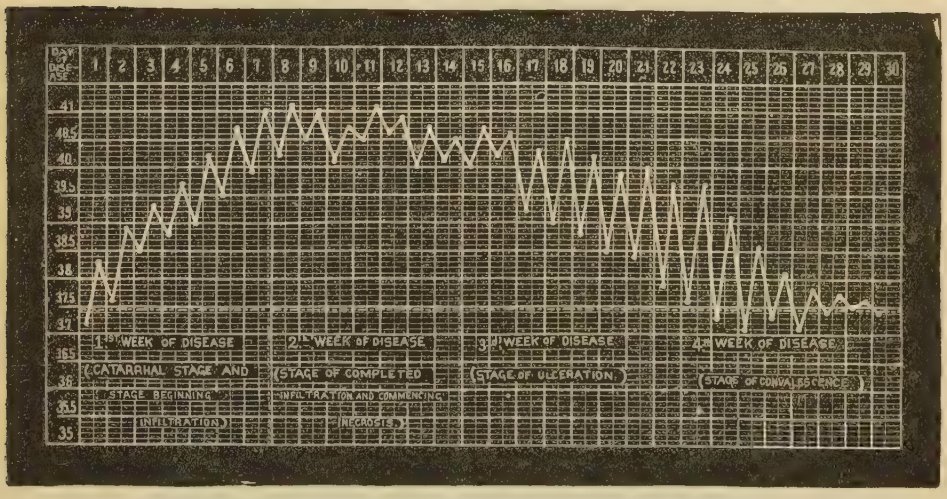
The prodromal stage generally lasts several days, more rarely a few weeks or only a few hours. The patients complain of general malaise, anorexia, restless sleep, inability to work mentally or physically. Not infrequently there are shooting pains in the muscles, generally in the legs, more rarely in the back.

The disease proper generally begins with repeated chilly sensations, more rarely with a single chill. As a rule, this is followed by rapid ele-

vation of temperature. The manifest symptoms depend chiefly on the anatomical changes in the intestines.

Typhoid fever is almost always associated with increased bodily temperature, and the curve is so characteristic as to enable us to make a diagnosis from it in doubtful cases. In the first week (catarrhal swelling and beginning medullary infiltration of the intestinal lymph follicles) the temperature rises gradually. Every night it is usually about 1° C. more than on the preceding night, but the next morning it is generally about 0.5° C. lower (vide Fig. 31). At the end of the first week the fever has generally reached its height, and then remains approximately continuous during the second week (completed medullary infiltration and beginning ulceration of the follicles). In the third week greater variations of temperature occur, the type of fever becoming re-

FIG. 31.



Typical temperature curve in moderately severe typhoid fever.

mittent. Gradual defervescence occurs during the fourth week (completion of ulceration, and cicatrization). The variations between the morning and evening temperatures are often very considerable. The inverse type is sometimes observed, *i. e.*, exacerbations in the morning, remissions in the evening.

The elevation of the bodily temperature is attended with other febrile symptoms, viz. acceleration of the pulse, diminished appetite, increased thirst, and diminished diuresis.

At the beginning of the first week, the tongue is sticky, moist, and covered with a thick, gray, grayish-yellow, or grayish-brown coating (desquamated epithelium, débris of food, and schizomycetes). In the second half of the first week, the dryness of the tongue increases, and its edges, as far as the tip, become unusually clean, and almost brick red. As the second week approaches, the coating is gradually exfoliated (beginning at the tip and gradually passing backward). This often occurs in the shape of a triangle, the apex of

which is situated at the tip of the tongue. The tongue is generally entirely clean in the first part of the second week. It is unusually dry and red, often rough and papular from swelling of the fungiform papillæ.

Towards the end of the first week, a peculiar eruption often appears in the shape of pale red, usually rounded patches which are slightly raised, and become entirely pale on pressure (typhoid roseola). As a rule, they appear first on the abdomen, but also upon the chest and back, where they are occasionally even more profuse than upon the abdomen. They are rarely found on the limbs, and then generally on the arms or thighs; they hardly ever occur on the face. The individual patches generally disappear in three to five days, but occasionally persist for more than a week. In the latter event, I have sometimes found that the paling of the patches is followed by slight desquamation of the epidermis. New crops of roseola may continue to return into the fourth week, and even into the period of convalescence. Their number varies according to the epidemics, but they may be so numerous as to remind us of measles. I have never seen typhoid fever without roseola, but some authors state that this occurs in rare cases. Fine vesicles sometimes form at the apices of the patches.

The abdomen is generally prominent, especially in its lower half, and in the ileo-cæcal region.

The ileo-cæcal region is almost always tender on pressure, and even unconscious patients distort the features when this region is palpated. More rarely there is tenderness on pressure over the epigastrium, or other parts of the abdomen. At the same time, a gurgling murmur is often felt in the ileo-cæcal region. This merely indicates the presence of fluid mixed with bubbles of gas beneath the compressing fingers, and while it is unusually frequent in typhoid fever, it is by no means characteristic. Percussion over this region generally furnishes a dull or dull tympanitic percussion sound.

In the second half of the first week, the spleen, as a rule, is found to be enlarged, and this increases considerably during the second week. It not infrequently attains two or three times the normal dimensions. If the patient is placed in the right diagonal position, and the fingers, without pressing in deeply, are placed between the anterior tips of the eleventh and twelfth ribs, the enlarged organ can almost always be felt. Sometimes its borders can only be felt indistinctly; in other cases, the tip and anterior portions can be distinctly felt. The organ is smooth, of peculiarly soft consistence, and not very rarely tender on pressure. In the third and fourth weeks the enlargement gradually diminishes.

Some authors believe that percussion of the spleen furnishes more important results than palpation. In our opinion, however, the possibility of feeling the spleen is one of the most constant symptoms of typhoid fever, provided that the examination is made in the manner mentioned above.

At the beginning of the disease the bowels are often constipated. Diarrhœa gradually sets in, and two to six stools are passed daily. They often look like pea soup; they are thin, of a light-yellow color, not infrequently have a biting, ammoniacal odor, and an alkaline reaction. On standing, they deposit a crumbly, partly flocculent sediment. The specific gravity is about 1.005, the proportion of solid constituents about 4 per cent. The typhoid stool is very poor in albuminoids. As recov-

ery progresses, the stools become more consistent and scanty, and finally normal.

Microscopical examination of the stools shows intestinal epithelium, round cells, débris of food, fat cells and crystals, necrotic tissue from the mucous membrane, round and rod-shaped schizomycetes, and triple phosphates (vide Fig. 32). Pfeiffer reports that he has succeeded in cultivating typhoid bacilli from the stools.

Typhoid fever rarely runs the simple course indicated above, and its symptoms are unusually manifold.

Among the different varieties we will first refer to those in which the

FIG. 32.

Typhoid stool, containing round cells, débris of food, granular detritus, and crystals of triple phosphates. Enlarged 20 times.

affection of other organs is so prominent as to give rise to the liability of overlooking the typhoid fever. We refer chiefly to pneumotyphoid, renotyphoid, and meningotyphoid.

Pneumotyphoid often creates the impression of an ordinary but severe fibrinous pneumonia. But we will generally be struck by the unusual degree of impairment of consciousness, the unusually large size of the spleen, and the presence of roseola on the integument. It is sometimes found that, despite the occurrence of absorption, the critical defervescence of pneumonia remains absent and the hitherto concealed typhoid symptoms become more noticeable. Not infrequently, however, death occurs before the crisis can be expected. Some authors assume that in this variety of the disease infection does not take place through

the intestinal tract, but through the respiratory organs, so that the first and most violent morbid phenomena are noticed in the lungs.

The terms bronchotyphoid and laryngotyphoid are sometimes applied to cases which are characterized by violent symptoms of bronchitis or inflammation and ulceration of the laryngeal mucous membrane. We regard these terms as misnomers.

Renotyphoid is characterized by prominent urinary symptoms from the very beginning of the disease. The urine contains albumin, casts and blood. The kidneys show parenchymatous or interstitial changes, occasionally merely a condition of cloudy swelling. In these cases, there is danger of overlooking the typhoid fever and regarding the disease as nephritis.

In meningotyphoid (cerebral typhoid), the suspicion of meningitis is aroused by rigidity of the nape, marked impairment of consciousness, sometimes by temporary inequality of the pupils. I have often observed such cases in conditions of high fever, and the meningitic symptoms often disappeared as soon as the temperature was lowered. We suspect that these symptoms depend on hyperæmic and œdematous conditions of the meninges.

A second group of varieties of the disease may be designated, according to the severity of the symptoms, as mild typhoid. All or at least the majority of the symptoms are very mild. The patients sometimes feel so well that they go about their usual business (walking typhoid). Some of these individuals may be suddenly attacked by severe complications (perforation-peritonitis, intestinal hemorrhage). In other cases, the fever is very mild, and in rare cases entirely absent. In such mild cases, we may be struck by the slight degree of enlargement of the spleen, scanty roseola, and mildness of the intestinal symptoms.

A third variety of the disease is known as abortive typhoid. The disease sometimes lasts only a few days, sometimes it continues to the beginning of the third week. Such attacks often assume the character of an acute disease; they begin with a chill, followed by continued high fever, and quite rapid defervescence, accompanied with profuse sweating. In very rapid cases, fully developed medullary infiltration does not seem to develop, at all events necrosis and ulceration of the intestinal lymph follicles do not occur.

Bronchitis is one of the most frequent complications of typhoid fever, and some authors claim that it is merely a symptom of the disease, not a complication. In our opinion, this is not true, since we have treated a number of cases in which no signs of bronchitis were noticeable. Bronchitis is characterized objectively by rude vesicular breathing, sonorous and sibilant râles, more rarely by moist râles. As a rule, the symptoms are most marked in the posterior and lower portions of the lungs.

Eisenlohr described acute bronchial croup which appeared at the beginning of typhoid fever, and disappeared at the end of the second week.

Pulmonary complications are not infrequent. Patients who occupy the same position for a long time are apt to develop pulmonary hypostasis, characterized by more or less dulness (occasionally by a tympanitic note), feebleness, sometimes absence of vesicular breathing, and non-consonant moist râles. If the position of the patients is frequently

changed, it is often found that the dull parts become resonant. If this is not done, hypostatic pneumonia is apt to develop. The dulness then becomes more marked, bronchial breathing is heard and the râles become consonant. These complications are so much more apt to develop the higher the fever, the more profound the unconsciousness, the greater the impairment of the heart's action. Fibrinous pneumonia sometimes develops in quite a latent manner. Apparently spontaneous rise of temperature, accelerated respiration, cyanosis, and slight clouding of consciousness should always lead to careful examination of the lungs. This complication develops most frequently after the second week.

In several cases, I have observed "foreign body" pneumonia; lobular catarrhal pneumonia is not uncommon. Hemorrhagic infarctions are sometimes produced, secondary to thrombosis of the right auricle, or marantic thrombosis of the veins. This is sometimes followed by abscess Pulmonary gangrene may occur, but rather as a sequel. Embolism of the pulmonary artery may give rise to sudden death. Pleuritis is a rare complication.

The bronchial glands sometimes undergo suppuration. This may be followed by mediastinitis, which Fraentzel also observed after inflammation of the œsophagus and peri-œsophageal connective tissue; the pus may rupture into the œsophgus, bronchi, pleural or pericardial cavities.

Typhoid ulcers develop not infrequently in the larynx, sometimes as early as the second week of the disease. They often give rise to no symptoms and are found only after laryngoscopic examination. In other cases, the patients are hoarse and complain of pain in swallowing and on pressure over the larynx. The ulcers are so deep at times as to give rise to perichondritis and necrosis of the laryngeal cartilages, even to perforation of the laryngeal walls and cutaneous emphysema. The ulcers may also be the starting-point for fatal œdema of the glottis.

According to Dittrich, laryngeal perichondritis may also develop in typhoid fever independently of specific ulcerations. Diphtheritic and croupous laryngotracheal inflammation has been observed at times, usually associated with similar changes in the pharynx.

In Zurich I often observed purulent or hemorrhagic inflammation of goitres; in some cases death from suffocation occurred, despite tracheotomy, as the result of laryngeal stenosis.

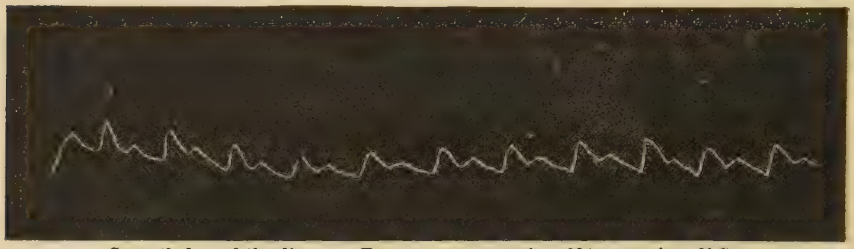
Nasal catarrh is a very frequent complication, accompanied by redness, swelling, and hence impermeability of the nose. Repeated epistaxis sometimes occurs in the prodromal stage or the first week of the disease, and may become dangerous from its profuseness. The patients often feel easier after the hemorrhage. Epistaxis occasionally does not develop until a later period, associated at times with signs of blood dissolution (petechiæ on the skin and mucous membranes, intestinal hemorrhage, hæmaturia, etc.), and is then an unfavorable prognostic sign.

The respiratory complications are sometimes nervous in character. Heckylei reports a case in which Cheyne-Stokes breathing and stuttering developed on the fourteenth day of the disease. In one of my cases, sudden hoarseness was produced in the third week by paralysis of the internal thyro-arytænoid and arytænoid muscles. In another case, complete unilateral paralysis of the recurrent laryngeal set in.

Pericarditis and endocarditis are rare complications on the part of the circulatory organs. There is often slight dilatation of the right side

of the heart, as in other febrile or exhausting diseases. The first (systolic) heart-sound is not infrequently soft and blowing, usually most markedly over the apex. Paralysis of the heart-muscle may set in

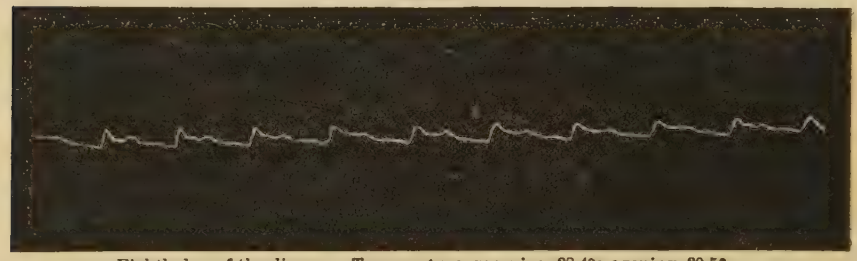
FIG. 33.



Seventh day of the disease. Temperature, morning, 38.5°; evening, 39.7°.

gradually or suddenly, at the height of the disease or during convalescence; in the former event, it is the result of the high fever, the severity of the infection, or both.

FIG. 34.



Eighth day of the disease. Temperature, morning, 38.4°; evening, 39.5°.

Cardiac thrombi may also form and, according as they affect the left or right side of the heart, will give rise to embolism of the spleen, kidneys, etc., or of the pulmonary artery.

Marantic thrombosis of the veins occurs rather as a sequel of typhoid fever. It is most frequent in the saphenous vein at its entrance

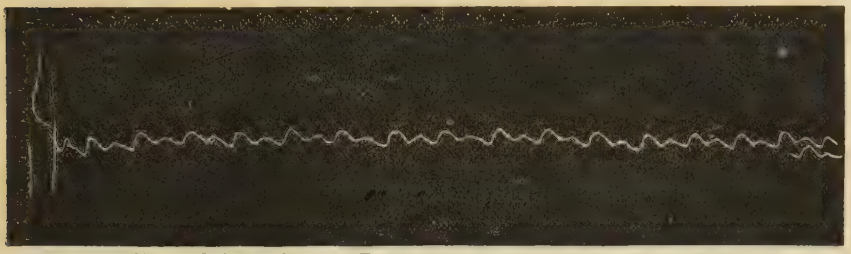
FIG. 35.

Eleventh day of the disease. Temperature, morning, 38.0°; evening, 38.6°.

into the crural vein, and in the latter itself. It is more frequent on the left side than on the right, and in men than in women. It is characterized by pain, numbness, coldness, and œdema of the limb; the throm-

bus is sometimes felt as a hard cord below Poupart's ligament. Palpation must be performed very cautiously, since particles are apt to be broken off and carried into the circulation as emboli. The thrombus

FIG. 36.

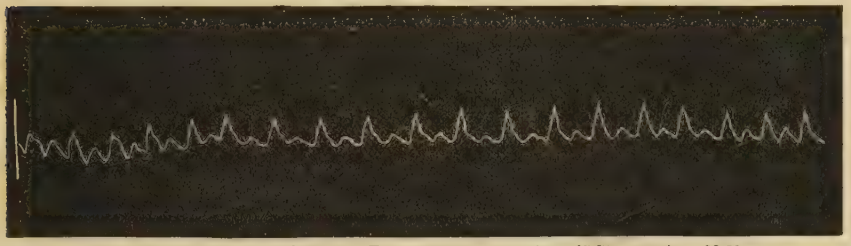


Sixteenth day of disease. Temperature, morning, 36.8°; evening, 38.4°.

sometimes extends into the inferior vena cava and then into the other crural vein.

Thrombosis in other localities is rarer. In a number of cases in Zurich, I observed thrombosis of the vena saphena magna from the knee to the middle of the

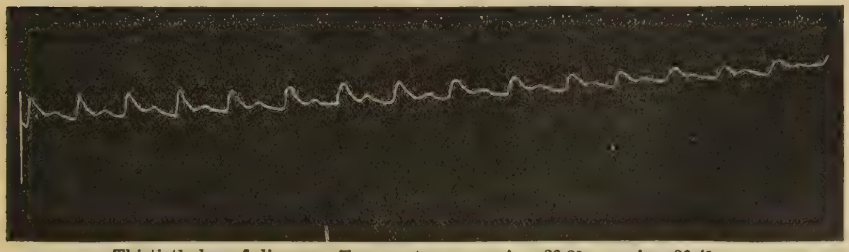
FIG. 37



Twenty-second day of disease. Temperature, morning, 35.7°; evening, 36.2°.

thigh, unattended with œdema, but followed by ulceration and discharge of a purulent, bloody fluid; termination in recovery. Cole described a thrombus of the innominate vein. I have observed several cases of periphlebitis in women suffering from varicose veins in the legs.

FIG. 38.



Thirtieth day of disease. Temperature, morning, 36.2°; evening, 36.4°.
Figs. 33–38.—Pulse curves from a typhoid fever patient, æt. 24 years.

The pulse is accelerated, but often less than would be anticipated from the height of the fever. As a rule, it varies from one hundred to one hundred and twenty beats a minute. A more rapid pulse indicates great

gravity. The pulse is sometimes unusually slow, without any ascertainable cause. It is generally full and soft, often distinctly dicrotic, or acquires the latter quality during the stage of recovery. If the hitherto regular pulse becomes irregular, small, and occasionally intermittent, there is danger of the occurrence of heart failure.

In Figs. 33–38 we furnish the sphygmographic tracings in a case of typhoid fever, taken on those days in which a change in the pulse was manifested. It is evident that during the course of the disease the pulse becomes more and more dicrotic, *i. e.*, the blood pressure gradually sinks.

No specific changes have been found in the blood. Occasionally there is a slight increase in the number of white blood-globules, or an unusual abundance of elementary granules, especially during convalescence. In one case, I found in the blood large granular cells which contained as many as seven red blood-globules.

The lips are generally dry ; rhagades are apt to form upon them, and the epithelial layers are exfoliated in part in the shape of yellowish and brownish scales. Hemorrhages are not infrequent. The blood dries into brownish-red or black crusts, and the lips look as if covered with soot. They are sometimes inflamed, thickened, and painful.

The gums are often swollen and red, and not infrequently covered with sordes. Abscess of the gums develops at times, and may form the starting-point for extensive ulcerations.

Fissures, sordes, and hemorrhages are often noticeable on the tongue, and its edges often contain the impressions of the teeth. These may give rise to extensive ulcerations. The tongue is often moved slowly and with difficulty from one side to another, and its movements are often trembling and uncertain. This has been attributed to various causes, such as general weakness, dryness, and stickiness of the tongue, but it must not be forgotten that cloudy swelling, fatty and waxy degeneration have been observed in the muscular fibres of the organ.

Catarrhal angina is an almost constant symptom in typhoid fever. White patches, consisting of swollen epithelium and fungi sometimes form upon the inflamed parts. Follicular angina may also develop. The condition becomes more serious when necrotic or diphtheritic changes appear in the pharynx, and extend, as they sometimes do, to the larynx and œsophagus. I have repeatedly observed that the uvula was almost entirely destroyed by necrosis, and that violent hemorrhages were thus produced. These lesions hardly ever develop before the end of the third week. They give rise to pain in the pharynx, and difficulty in swallowing. Some writers claim that there is occasionally a specific typhoid angina which causes extensive superficial losses of substance.

Parotitis develops in a few cases, sometimes even inflammatory changes in the other salivary glands. These may develop from the extension of catarrh of the mouth to Steno's duct and occlusion of the latter, or from typhoid changes which occur not infrequently in the parotid glands. If suppuration ensues, death may occur from exhaustion or pyæmia, or the pus burrows, erodes blood-vessels, destroys the facial nerve, ruptures into the external auditory canal, etc. These changes are late complications, and do not appear before the end of the third week of the disease.

Mosler states that only a few drops of saliva can be obtained from the parotid in typhoid fever, and that its reaction is acid.

In rare cases, sprue develops on the buccal mucous membrane and may even extend into the œsophagus.

Thirst is always increased, though apathetic patients do not ask for drink. The appetite is poor, but boulimia may be observed during convalescence. The taste in the mouth is generally said to be pasty, sometimes nauseous. Vomiting is not infrequent. Griesinger states that vomiting may be so obstinate during the prevalence of cholera that a diagnosis of the latter disease is apt to be made rather than of typhoid fever. Other patients suffer from an annoying feeling of nausea.

Catarrhal inflammation, necrosis, and ulceration, and sprue have been observed in the œsophagus. The inflammation sometimes extends to the peri-œsophageal tissue, and then to the mediastinum. In one case, Lindner observed uncontrollable œsophageal spasms as soon as an attempt was made to drink, during the third week of the disease. The autopsy revealed a gelatinous exudation on the surface of the brain, and trifling catarrh of the pharynx and œsophagus.

Hæmatemesis has been described in a few cases, and is secondary to round gastric ulcer or to excessive congestion of the gastric mucous membrane.

Serious intestinal complications are not infrequent. Thus, the diarrhœa may become very severe (more than twenty evacuations daily), and is often attended with tenesmus. The fæces are often passed in bed. Such conditions may result in fatal prostration. Erythema of the anal and sacral region may develop in such cases, and may be the starting-point of severe inflammations of the skin, gangrene, and bed-sores. In some cases, on the other hand, constipation continues during the entire course of the disease. It may then happen that the thick and dry fæces irritate the intestinal mucous membrane and give rise to intestinal hemorrhage or perforation.

Intestinal hemorrhage must be regarded as a serious complication, despite the fact that immediately afterwards we may find that the temperature falls temporarily and even becomes subnormal, consciousness becomes clearer, and the patients feel relieved. If the hemorrhage is very profuse, it may prove fatal at once, or it may be checked at first, but soon recurs, and terminates fatally. Soon after the beginning of the hemorrhage, the pulse often becomes very dicrotic, and I have also repeatedly observed slight temporary œdema of the malleoli. In one case of relapsing intestinal hemorrhage, Traube observed extensive œdema and death from œdema of the glottis. Rapid diminution in the size of the spleen occurs not infrequently after the hemorrhage.

The most frequent cause of the hemorrhages is the erosion of vessels following exfoliation of the necrotic scurf of the intestinal ulcers. The bleeding vessel is not always easily found at the autopsy. We should specially examine those ulcers which are situated nearest to the first part of the bloody intestinal contents. Or the mesenteric artery is injected by means of a canula, and the point of escape of the injection mass sought for. In rare cases, the hemorrhage is the result of excessive congestion of the mucous membrane (so-called capillary hemorrhage). From a clinical standpoint, we distinguish latent and manifest intestinal hemorrhage. In the former, death may occur before any blood escapes from the anus. The occurrence of latent intestinal hemorrhage may be assumed if great pallor of the face and body suddenly sets in, the face looks ghastly, the limbs are cold and covered with clammy sweat, the pulse becomes small and imperceptible; at the same time the abdomen is distended, and in

certain parts, corresponding to the site of hemorrhage, there is a feeling of increased resistance on palpation, and a dull tympanitic percussion note. Blood which is passed in the stools is usually dark-red, sometimes blackish-brown, usually coagulated, more rarely fluid. Several litres may be discharged, and death has been known to occur in an hour. Intestinal hemorrhage is unusually frequent in certain epidemics. It generally occurs spontaneously, in rare cases it is produced by constipation or incautious movements.

Still greater dangers arise if perforation of the intestines takes place, followed by perforation-peritonitis. This may take place if the ulcers have extended deeply, so that the thin portion of the intestine is ruptured from straining at stool, coughing, vomiting, from the ingestion of indigestible articles, etc. Ascarides in the intestines may favor perforation, although their presence in the peritoneal cavity may be owing simply to the tendency of the worms to pass through narrow openings.

Perforation does not occur, as a rule, before the third week, occasionally as late as the ninth or tenth week. The patients often complain of intolerable abdominal pain at the time of perforation. They grow pale very rapidly, often feel as cold as ice, and the pulse is small and rapid; the abdomen is distended and tender on pressure; hepatic and splenic dulness disappear when gas enters the peritoneal cavity and separates the organs from the abdominal walls. Dulness soon sets in, corresponding to peritonitic exudation. Vomiting of grass-green, watery, or porridge-like masses is not infrequent. In two cases I observed stercoraceous vomiting without intestinal occlusion. The temperature generally falls below the normal, and the sensorium often becomes clearer. In other cases, the bodily temperature rises. Death follows not infrequently in a few hours, usually in twenty-four to ninety-six hours. Recovery is exceptional.

In one case, Tschudnowsky observed amphoric breathing over the abdomen, synchronously with the movements of respiration. It appeared to be caused by escape of gas through the site of perforation into the peritoneal cavity, owing to rhythmical compression of the loops of intestines. Intestinal perforation is more frequent in men than in women, generally affects the small intestine, more rarely the vermiform process or colon. There may be several perforations at the same time.

Hepatic and splenic dulness does not disappear if the organs are fixed in their position by old peritonitic adhesions.

The perforation is sometimes preceded by adhesions of the loops of intestines and inflammatory changes in the peritoneum. Free perforation does not take place under such circumstances, the changes run a slower course, and are apt to be overlooked.

Typhoid fever may also be complicated by simple peritonitis, either circumscribed or diffuse. These changes start from inflammation of the serous membrane overlying the intestinal ulcerations.

In some cases, necrotic or diphtheritic changes develop upon the mucous membrane of the large intestine. In one case, Scott observed intestinal invagination which terminated in recovery after exfoliation of a piece of the gut six inches in length.

Excessive tympanites sometimes induces great danger, inasmuch as the compression of the heart and lungs may cause suffocation.

Excessive increase in the size of the spleen leads to rupture of that organ in very rare cases. Splenic infarctions develop much more

frequently; they are often secondary to thrombi of the left heart, more rarely to recent endocarditic deposits. These may induce peritonitic symptoms, or lead to suppuration and the formation of a splenic abscess, which ruptures into the peritoneal cavity or other organs.

The liver is very often swollen and slightly painful. Abscess or acute yellow atrophy of the liver develops in rare cases. As in many other febrile diseases, slight jaundice of the sclera is not at all uncommon. Hæmatogenous icterus, associated with hæmoglobinuria, appears to have been present in a case reported by Immermann.

Necrotic or diphtheritic processes sometimes form on the wall of the gall-bladder, and may give rise to perforation, with subsequent diffuse peritonitis.

The urinary changes depend entirely on the fever.

Its amount is diminished, reaction very acid, specific gravity increased; a brick-red sediment of acid urate of soda is found not infrequently. The urea is increased, especially during the first week, but diminishes during convalescence. If the temperature is reduced by treatment, the amount of urea generally increases. The quantity of kreatinin and ammonia is increased. Salkowsky showed that the amount of potassium sinks very considerably upon the occurrence of convalescence.

Leucin and tyrosin have been found in the urine, occasionally bile pigment, although jaundice is not present. Hæmoglobinuria has been reported a number of times. Gerhardt observed peptonuria.

Albuminuria is not uncommon during the course of typhoid fever. It is generally pyrexial and does not appear until the end of the first week. Acute parenchymatous nephritis develops occasionally; the amount of albumin is then larger, and the sediment contains casts and red blood-globules. Death from uræmia may take place during the course of typhoid fever. Wedge-shaped infarctions are found occasionally in the kidneys.

The symptoms of mild cystitis or pyelitis soon become noticeable. In unconscious patients, the bladder is often distended and may reach to the umbilicus.

Diphtheritic changes develop very rarely on the mucous membrane of the urinary passages.

Menstruation often becomes irregular, and amenorrhœa is almost always noticed for two or three months after recovery from typhoid fever.

Orchitis and epididymitis have been observed in men. Phlegmonous inflammation and gangrene of the genitals may occur in both sexes, and may be the cause of death.

The functions of the nervous system are always more or less impaired in this disease. Headache begins very early. The pain is either diffuse or localized in certain nerve tracts on one side of the head. In addition, there is often hyperæsthesia. In some cases, there is very early hyperæsthesia or anæsthesia of the entire skin or certain parts; partial or general convulsions and trismus have also been observed. Consciousness is soon impaired. The patients feel incapable of thinking, complain of dulness in the head, they are sleepless and restless at night, somnolent during the day. At first delirium is apt to set in just before falling asleep. Gradually the patients become more and more apathetic, mutter to themselves, or move the lips and tongue in a trembling manner. If they are still able to answer questions, the words are spoken in

a jactitating, tremulous manner. The patients move the hands restlessly, pick at the bed clothes, and often suffer from involuntary muscular twitchings; movements of the tendons of the forearms (subsultus tendinum) are especially frequent. Chorea has been observed at the height of the disease. When recovery begins, consciousness slowly clears and the patients awake as if from a long sleep. The memory of events which occurred shortly before and during the illness is lost, that of events long past is unaffected. In some cases, the patients are apathetic but quiet, must be fed artifically, and do not seek to satisfy their natural wants. In other cases, the patients are delirious, boisterous, violent, and make attempts at flight or suicide. These are the patients who, in an unguarded moment, rush to the window to jump to the street, escape in their night-gown, throw themselves into the river, etc. Such conditions not infrequently assume a decidedly maniacal character. They may develop even during the prodromal period. In some cases, delusions develop during the course of typhoid fever, and may persist for a long time during convalescence.

In one of my cases, I observed, during the fourth week of the disease, the sudden onset of apoplexy with right hemiplegia and aphasia, but these symptoms gradually diminished, and disappeared at the end of two weeks. The tendon reflexes may be increased, unchanged, or diminished.

Among the organs of special sense, the ear is often affected, the majority of patients complaining of impairment of hearing and ringing in the ears. These symptoms depend in great part on tubal catarrh which has extended from the pharynx. In several cases, Hoffmann found purulent otitis media, with and without perforation of the drum membrane. The pus may extend along the Fallopian canal to the cranial cavity and there produce secondary purulent meningitis, or it gives rise to compression and paralysis of the facial nerve.

The following ocular complications have been described: ulcers of the cornea, mydriasis, paresis of accommodation, transitory and permanent amaurosis (optic atrophy). Galezowsky mentions optic neuritis and perineuritis.

In the beginning of the disease and during the stage of continued fever, the skin is usually dry. Sweats occur when the fever becomes remittent and miliaria are often observed at the same time. In some cases, sweats are observed from the beginning. Petechiæ sometimes develop upon roseolæ or independently of them; their significance is grave if there are other evidences of blood dissolution or a hemorrhagic diathesis (hemorrhages from the gums, nose, genitals, intestines, etc.). Indistinct, bluish-red patches which do not disappear on pressure (pelioma typhosum) are found occasionally on the trunk and limbs, but possess no special significance. Herpes labialis is extremely rare, and its presence is, to a certain extent, opposed to the diagnosis of typhoid fever. Diffuse erythema is occasionally observed, most frequently upon the chest and abdomen, more rarely on the limbs, and then particularly on the extensor surfaces. We must be on our guard against mistaking this for scarlatina. Decubitus is a very important complication. It is seen most frequently upon the sacrum, next upon the trochanters, malleoli, elbows, occiput, etc. It may cause erosion of the sacrum and extend to the meninges, or spread from the trochanter into the hip-joint. In many cases, it is the result of bad nursing. Constant rest in one

position and folds in the bedding will produce it. But in some cases it forms despite the best of care, and is then the result of trophic disturbances of the skin, partly from the accumulation of abnormal products of disassimilation in the blood, partly from the enfeebled circulation in the cutaneous vessels. Erysipelas occasionally develops without apparent cause; it generally begins near the nose, but not infrequently extends to the neck and chest.

Muscular pains are a frequent complication and are attributed by many to the anatomical lesions found in the muscles. Hemorrhages, abscess, and rupture of the muscles are occasionally produced. The rectus abdominis is affected most frequently, especially in certain epidemics.

The weight of the body diminishes to a not inconsiderable extent, less in children than in adults. During convalescence, the weight of the body is sometimes restored very rapidly, in other patients this takes place very slowly.

The complications of typhoid fever often pass imperceptibly into the sequelæ. Furuncles and abscesses may develop in the skin, are often of pyæmic origin, and sometimes cause a fatal termination by the loss of vitality entailed. Petechiæ or œdema of the lower limbs, pains or cramps in the muscles sometimes appear after the first attempts at standing. The lymphatic glands may also be affected and give rise to slow processes of suppuration.

In one case, Litten observed extensive pigmentation of the skin in the shape of blackish patches which he attributed to an affection of the sympathetic; in another case, he observed an eruption like erythema.

Necrosis and spontaneous gangrene sometimes develop in peripheral portions of the body, such as the nose, genitalia, toes, etc. Arterial thrombosis has been found in a number of these cases. In a case of gangrene of the female genitals, Eppinger found the finer vessels filled with thrombi of micrococci. Noma may also occur.

Inflammatory changes may also occur in the bones. Mensel reports extensive necrosis of the skull from thrombosis of a branch of the middle meningeal artery. According to Paget, post-typhoid periostitis occurs most frequently upon the tibia, next upon the femur, ulna, and parietal bone. In exceptional cases, the affection is unilateral.

Suppuration of the joints followed by anklyosis has been described in a number of cases. In a few cases, I have observed temporary painful swelling of the joints at the height of the disease.

Defluvium capillitii is an almost constant sequel of typhoid fever, and may be so extensive as to produce almost complete baldness. This condition disappears after a while.

Valvular lesions of the heart and Basedow's disease have been observed as sequelæ. Nothnagel mentions neurosis vasomotoria.

Pulmonary abscess and gangrene may also be included among the sequelæ. Chronic pulmonary phthisis and miliary tuberculosis develop occasionally.

Necrosis of the laryngeal cartilage may be left over. There may also be functional paralysis of certain muscles of the vocal cords, the dilators of the glottis being attacked with relative frequency.

The stomach and intestines sometimes remain sensitive for life, or the evacuations from the bowels remain thin and frequent for life. Suppuration of the perirectal cellular tissue (periproctitis) occurs in rare

cases. Slight enlargement of the spleen persists permanently in some cases.

Chronic Bright's disease is rare as a sequel of typhoid fever. Polyuria often sets in during convalescence.

Nervous disturbances are often left over. Many patients exhibit, for a long time, lack of desire for mental activity, indifference, and feebleness of memory. Such individuals may never recover their previous mental vigor. Insanity develops more rarely and, according to Nasse, previous insanity sometimes disappears after typhoid fever. Meningitis, thrombosis of the sinuses or middle cerebral artery, hemorrhages into the meninges or cerebral parenchyma, and purely functional nervous disturbances are occasionally observed. Typhoid fever, like other infectious diseases, may also be followed by paralysis of individual nerves and muscles, which, in some cases at least, are the result of neuritis. Ataxia, chorea, tremor, paraplegia, and sensory disturbances have also been observed.

In favorable cases, the duration of typhoid fever, including convalescence, may be given as eight weeks, though the disease often lasts much longer.

Relapses occur occasionally, and their frequency appears to depend on the character of the epidemic. Among other causes mentioned are: too early ingestion of solid food, excitement, and getting out of bed too early, but the importance of these factors has been overestimated. There is no doubt that relapses may occur despite every precaution, and it is a question whether it depends upon a new infection, or whether previously present typhoid virus had been permitted to unfold its pernicious properties. The latter view seems to us more plausible, since Gerhardt has shown that there is danger of a relapse if, after the disappearance of the fever, the enlargement of the spleen does not diminish; moreover, typhoid fever is one of those infectious diseases in which infection generally occurs only once.

According to Ebstein, relapses are liable to occur in feeble constitutions. Immermann thinks that their frequency is increased by the antipyretic method of treatment. A relapse is less apt to occur the more severe the course of the disease has been. It generally runs a shorter and milder course than the first attack and is more rarely followed by sequelæ. The spleen again enlarges; roseola is almost always present, sometimes more abundantly than during the first attack. The relapse may begin with a sudden chill and elevation of temperature, or the bodily temperature rises slowly, after it has been apyrexial for days. It generally terminates in recovery. In Leipzig, the frequency of relapses varied in different epidemics from 2.4 to 11.3 per cent.

True relapses must be distinguished from the exacerbations which may occur before the disease has run its course, and from the so-called after-fever, *i. e.*, brief rises of temperature during the period of convalescence.

IV. Diagnosis.—The diagnosis of typhoid fever is usually easy, but it is sometimes mistaken for typhus, measles, scarlatina, variola, pneumonia, meningitis, nephritis, miliary tuberculosis, septic endocarditis, florid syphilis, and febrile gastro-enteritis.

In the differential diagnosis between typhus and typhoid fever, chief stress must be laid on the abundance and character of the eruption, the more sudden onset and critical termination in typhus, and the shorter duration of the latter disease.

In measles, the face is particularly affected by the eruption.

Scarlatina begins more acutely and leads to desquamation of the skin.

Typhoid fever may be mistaken for variola in the prodromal stage, but in the latter pains in the loins predominate.

In doubtful cases, the diagnosis of pneumonia is favored, apart from the pneumonic sputum, by the presence of herpes labialis.

In meningitis, there are rigidity of the nape, paralytic and irritative symptoms in the limbs, and changes in the fundus of the eye.

In miliary tuberculosis, special attention should be paid to the fundus of the eye (choroideal tubercles).

Septic endocarditis requires careful examination of the heart for murmurs, and search for embolic processes.

Florid syphilis is associated with changes on the genitals.

Acute gastro-enteritis runs a shorter course than typhoid fever, and the febrile movement is generally less.

V. Prognosis.—The prognosis is always serious, but the mortality varies remarkably in different epidemics. The disease runs a more favorable course in children than in adults, because true ulceration of the intestines rarely occurs in the former, and complications are much less frequent. The danger is so much greater the higher and the more protracted the fever.

Severe clinical symptoms do not always correspond to extensive intestinal changes and vice versa, and the severity of the general infection is an essential feature in prognosis.

VI. Treatment.—Prophylaxis is an important element in treatment. The stools and clothing of typhoid fever patients must be thoroughly disinfected. When water-closets are cleaned, care should be taken that their contents be not allowed to enter adjacent streams, etc. Care must be taken to secure good drinking water and to clean the soil of excrementitious matters as much as possible by suitable canalization.

When typhoid fever breaks out in a house, we should endeavor to ascertain its cause in order to protect the other inhabitants. The safest plan is removal from the infected house. The treatment of typhoid fever itself is purely symptomatic.

The patient's room should be large, airy, and kept constantly at a temperature of 15° R. It should be aired several times a day. Too bright light should be avoided.

The bedding must be carefully smoothed, and this must be seen to several times a day. The position of the body, especially if the patient is apathetic, should be changed every hour, in order to avoid excessive pressure on certain parts of the body and the development of hypostasis in the lungs. The back may be rubbed every morning and evening with lukewarm water, to which alcohol, vinegar, or eau de cologne has been added. If possible the patient should have a separate bed for day and night use. He may drink water, to each glass of which a teaspoonful of brandy or two or three tablespoonfuls of white or red wine may be added.

So long as fever is present, fluid diet alone should be given: milk, soup, oatmeal or barley water, a soft-boiled egg, wine, beer, coffee au lait, etc. When the fever subsides, we may gradually return to solid food, beginning with a few spoonfuls of farina or mashed potato, then adding beef-tea or a pigeon which has been thoroughly boiled and strained through a cloth, then scraped raw meat, etc.

The bladder should be emptied at regular intervals, and an evacuation from the bowels secured at least every other day; the bed-pan must be used and straining avoided. If necessary, a mild laxative may be given (calomel, gr. vij. at one dose).

Finally, the patient should receive two lukewarm baths (26° R.) daily, one at 9 A.M., the other at 4 P.M. They must be kept under constant supervision, especially if they are delirious.

These measures are entirely sufficient in many cases, though we are often forced to prescribe a placebo.

In not a few cases, antipyreties are indicated on account of the severity of the fever. Such remedies are only indicated when the temperature remains constantly or for a long time at an unusual height (above 40.5° C.); or if the patient is old, feeble, or a drunkard, or in pregnant women, in whom the fever may produce abortion. Opinions differ as to whether the antipyretic should be administered in the evening, during the night, or in the morning (9–11 A.M.). We prefer the latter method.

In some cases, antipyretics produce increase of the bodily temperature. Different patients react differently to this or that plan of treatment.

The antipyretics include cold baths, antipyrin, quinine, salicylic acid, benzoic acid, kairin, thallin, digitalis, and veratrine.

The cold-water treatment, when carried out strictly, consists of the hourly administration of a bath whenever the axillary temperature exceeds 39.5° C. The temperature of the bath should be 15° R.; its average duration ten minutes. Cold frictions, cold pack, and the ice-bag have also been recommended, but their antipyretic action is much less vigorous than that of cold baths.

Riess recommends protracted (twelve to twenty-four hours) lukewarm baths (28–30° C.).

The treatment with antipyrin is certain and more convenient than the use of cold baths, but it does not exert such an invigorating effect upon the patient. We give, by enema, a single dose of ʒ i.–iss. dissolved in ℥ ij. of lukewarm water. The remedy may be continued, despite the production of antipyrin eruption. During the action of the drug, wine should be given in large quantities to prevent the occurrence of collapse.

Treatment with quinine is less certain. In adults, a complete antipyretic effect will not be attained with less than gr. xxx. of hydrochlorate of quinia. We give gr. vij. in a wafer every half-hour until gr. xxx. have been taken. When mixed with starch and lukewarm water, it may be given by enema, but sometimes causes disagreeable tenesmus. If the stomach and rectum are very irritable, it may be given subcutaneously (℞ Quiniæ hydrochlor., glycerin., aq. destil., āā. ʒ i. M. D. S. One syringeful, warmed).

Salicylic acid or salicylate of soda, gr. vij., may be given in wafer or mixed with succus liquirit. in a tablespoonful of claret, every fifteen minutes, until six doses have been taken. If necessary, twice this amount may be given. Benzoate of soda, which is much less effective, may be given in the same way.

Kairin lowers the temperature, but produces very disagreeable incidental effects. The patients often become cyanotic, ice-cold, are covered with cold, clammy sweat, the pulse becomes small and hardly perceptible, and there may be difficulty in respiration; in addition, violent chills occur when the temperature again rises; in addition, it is an inconvenient remedy, inasmuch as it necessitates hourly measurements of temperature. It is given in doses of gr. vij.–xv. every hour until the temperature has become apyrexial, and is again administered when the temperature reaches 38.0° C.

Thallin is also a certain antipyretic, but produces similar, though less severe, disagreeable incidental effects. In one of my cases, its administration was followed by marked albuminuria. Gr. iv. are given every hour until the temperature becomes normal.

Digitalis and veratrine have very little antipyretic action.

Profound unconsciousness and delirium disappear not infrequently under antipyretic treatment; in addition, an ice-bag may be applied to the head, and large amounts of stimulants given if the patient is very feeble.

If the mouth is very dry, it should be wiped every two hours with a piece of wet linen; dry, fissured lips are anointed with cold cream or vaseline.

In conditions of cardiac weakness, large doses of alcohol must be given from the start. If necessary, coffee, tea, champagne, camphor (subcutaneously), etc., must be resorted to. Under such circumstances, the patient must not be allowed to sit up in bed.

Expectorants are indicated in extensive bronchitis; in pulmonary hypostasis, the position of the patient should be frequently changed.

If there is frequent and exhausting diarrhœa, we may order Dover's powder (gr. iij. every two hours) or ℞ Bismuth. subnitrat., gr. vij.; pulv. opii, gr. ½, every two hours.

In severe tympanites, the abdomen should be rubbed every three hours with turpentine, then covered with lukewarm compresses, and an enema of lukewarm water administered. In our opinion, puncture of the intestines with a fine trocar is dangerous, because the intestinal contents may escape through the opening and set up perforation-peritonitis.

In intestinal hemorrhage, an ice-bag is applied to the ileo-cæcal region or to the part in which the origin of the hemorrhage is suspected, an injection of ergotin made in this region, and bismuth and opium given internally. Liquor ferri sesquichlorat. (gtt. v.–x. every two hours) has been strongly recommended by many writers.

If the formation of bed-sores threatens, the patient should be placed on an air cushion or water-bed, and the skin covered with smoothly applied adhesive plaster. When the bed-sores are very extensive, the patient should be placed in a permanent water-bath.

4. *Dysentery.*

I. Etiology.—Dysentery is an inflammatory affection of the mucous membrane of the large intestine, which is generally epidemic and depends on a specific infection. The inflammation may be catarrhal, purulent, or, in an anatomical sense, diphtheritic.

It is endemic in many tropical countries, for example, East India, Ceylon, Java, etc., and also in the Balkan Peninsula and in Spain and Portugal.

In the temperate zones, epidemics may develop from imported cases, crowded institutions being especially apt to suffer.

Like typhoid and typhus fever, dysentery is one of the most dangerous diseases during wars, and also puts an end to many an exploring expedition in the tropics.

The outbreak of epidemics is favored by certain circumstances, for example, climate. The spread of the disease is favored by hot seasons, and winter epidemics occur much less frequently than those in the sum-

mer. Hot days followed by very cold nights are regarded as especially dangerous. This is also true of protracted rains and absence of winds. The spread of the disease also seems to be favored by marshy lowlands. Dysentery in armies sometimes disappears suddenly upon breaking camp. Or one army suffers extremely from the disease, while another, encamped in close proximity to the former, escapes entirely. Malaria and dysentery are often active at the same time, especially in the tropics. In not a few cases, the patient is attacked at the same time by intermittent fever and dysentery, although it is erroneous to assume that the germs of the one disease may be converted into those of the other. In the Weimar epidemic of 1868, Pfeifer noticed that the first cases occurred in those houses in which the first cases of typhoid fever and cholera had also been observed. It has also been found that in successive epidemics the disease always started in certain houses. Overcrowding, bad air, dirty streets and houses favor the development of the disease. This is also true of excesses of all kinds, errors of diet and colds. Psychical conditions also appear to be significant. At least, Seitz states that, during the Franco-Prussian war, the disease appeared particularly among the despondent French prisoners.

The disease never develops autochthonously. It is always the result of the introduction of a specific dysentery germ, although this cannot be demonstrated in all cases. Certain cases may run such a mild course that their real character is unrecognized, and such cases are especially dangerous as regards the spread of the disease.

The virus is contained in the stools. It probably proliferates there, so that the infective power of the stool increases when the fæces stagnate. Bed pans, syringes, and water closets which have been used by patients suffering from dysentery and have not been thoroughly disinfected, may serve to convey the contagion. This is also true of articles of clothing, sources of water supply which communicate with water closets, and food. It is said that the virus sometimes retains its power of infection for ten years.

The nurses and other patients are very often attacked in hospitals, if the dysentery patients are placed in general wards. These patients should, therefore, always be isolated.

In several of my cases. the patients were attacked a number of times.

Nothing is known concerning the nature of the virus of dysentery, and even enthusiastic adherents of the germ theory are extremely reserved with regard to the mycotic character of the disease. The unknown virus is probably absorbed through the anus, mouth, or naso-pharyngeal cavity.

II. Symptoms.—The duration of the period of incubation varies from three to eight days.

Prodromata are absent in many cases, but some patients complain of anorexia, coated tongue, pressure in the epigastric region, colicky pains in the abdomen, and irregular evacuations. These symptoms may last more than a week.

In a few cases, the disease begins suddenly with a chill or repeated chilly sensations. Intestinal disturbances are often present from the beginning. The specific symptoms consist of frequent evacuations of peculiar composition, tenesmus, tormina, and borborygmi, tenderness on pressure, and pain in the left iliac region.

Twenty to thirty evacuations from the bowels are not infrequently passed daily, sometimes as many as sixty, one hundred, or even more.

The daily amount of the stools generally varies from ℥ xxv.–xxxv. Sometimes only three or four drachms are passed at a time.

The evacuations consist of fæces, mucus, pus, and blood in varying proportions. At first the intestinal contents are passed in a fluid condition. Masses of mucus then appear, partly adherent to fæces, partly separate. They often contain small, vitreous clumps, looking like swollen sago. These are not always composed of mucus. Virchow found that swollen starch granules may assume a similar appearance. After the fæcal contents of the bowels have been discharged, purely mucous masses may be evacuated.

It is supposed that the secretion of the mucous glands is pressed out upon the mucous membrane on account of follicular ulcerations, then assumes a round shape, and is excreted.

In advanced cases, more and more pus is evacuated, and is readily recognized from its color and opacity. It appears not infrequently in the shape of small flocculi or larger shreds. The stool sometimes contains hardly anything but pure pus, especially if abscesses have formed in the submucous tissue of the intestines.

In many cases, the stools present a bloody appearance, and even almost purely mucous stools generally contain dots and streaks of blood. The blood is often present in such amounts, and is so intimately mingled with the other constituents, that the stool has a uniform bloody color, in which the mucous and purulent flocculi are easily recognized. The intimate mixture of mucus and blood often gives to the stool the appearance of the rusty sputum of fibrinous pneumonia.

The stool sometimes consists entirely of blood. This occurs when the congestion of the mucous membrane is unusually active at the beginning of the disease, or in the later stages if ulcers have formed and eroded the vessels.

The stool sometimes has a cadaverous odor and a blackish color, and contains exfoliated shreds of mucous membrane. Such cases are known as putrid or gangrenous dysentery, and generally run a fatal course.

The evacuation sometimes loses the fæcal odor and occasionally smells somewhat like semen. Its reaction is generally alkaline or neutral, rarely acid. It contains a large amount of albumin, and this explains the fact that the patients rapidly grow pale and often assume a cachectic appearance.

Under the microscope the passages are found to contain round cells, red blood-globules, more or less altered epithelium, drops of fat, crystals of triple phosphates, bile pigment, débris of food, and innumerable schizomycetes of various shapes.

The majority of patients complain, before the evacuations, of borborygmus, colicky pains (tormina), and very soon of violent, almost uncontrollable tenesmus. The pain becomes especially severe when the stool passes the anal opening, and may be so intense that syncope occurs, or the patients grow pale and cold, the skin becomes clammy, and the pulse imperceptible. The anus is often retracted, and inspection reveals spasmodic twitchings of the sphincter. In men, the cremaster is often strongly contracted, and the left testicle drawn upwards against the inguinal ring. After dysentery has lasted for some, the anus and sur-

rounding parts become red, and erythema is apt to develop. Prolapsus ani may be produced by the violent tenesmus. After a time, a paralytic condition of the sphincter sets in, the anus remains open, and the contents of the rectum trickle out in a constant stream.

In the beginning, the abdomen is occasionally somewhat distended, in advanced stages it is sunken. The left iliac fossa is generally tender on pressure, and often somewhat resistant. If the dysenteric process is very extensive, tenderness may be felt along the entire colon and even over a part of the small intestine. Gurgling is often felt on gentle pressure over the affected parts, while a dull or dull tympanitic sound is heard on percussion.

The urine is generally scanty and saturated, and often contains albumin. Tenesmus of the bladder is often noticeable. Slight tenderness on pressure over the stomach, vomiting, and singultus are occasionally observed. The tongue presents a gray or yellowish coating. Thirst is generally increased, the appetite lost.

Uffelman investigated the digestive secretions in a dysentery patient who also suffered from a biliary fistula. He arrived at the following results: the saliva diminishes considerably in amount in severe cases and its reaction often becomes acid. At the same time it loses its sugar-producing property more or less completely, and does not contain rhodankalium. It is poor in salivary corpuscles, but contains numerous epithelium cells, granular detritus, and fungi. The gastric juice becomes more acid, and, in mild cases, will convert albumin into peptones. In severe cases, it becomes alkaline and loses its peptonizing property. The biliary secretion ceases in severe cases, and the bile produced in the beginning of convalescence does not acquire the normal color for four or five days.

The bodily temperature is sometimes unchanged; in other cases irregular; usually remittent febrile movement sets in. Putrid dysentery is not infrequently associated with typhoid symptoms: fever, clouded sensorium, delirium, dry fuliginous tongue and lips, acceleration of pulse out of proportion to the bodily temperature. The patients are rapidly prostrated and die in collapse (adynamic dysentery).

The dysenteric symptoms often last one to four weeks before gradual improvement takes place. The tenesmus and tormina gradually grow less, the stools assume more and more of a fæcal character. But the mere evacuation of fæcal masses, if unattended with other signs of improvement, should not induce us to prophesy recovery, since scybala are sometimes retained (probably on account of circumscribed spasm of the intestinal muscular coat) and are suddenly expelled after the lumen of the gut has become pervious.

Dysentery may also run a chronic course. The patients have purulent evacuations for months, and finally die of marasmus. Chronic ulcers and submucous abscesses or fistulæ of the intestines are generally found in such cases. Death from collapse is not rare, even in acute and subacute cases.

Complications and sequelæ are not uncommon in dysentry. Roseola has been noticed, but possesses no significance. Valentiner observed extensive dilatation of the cutaneous vessels and atrophy of the skin after dysentery. The intestinal changes sometimes extend to the peritoneum, and produce circumscribed or diffuse peritonitis. Even perforation-peritonitis may occur. The perirectal cellular tissue sometimes undergoes inflammation and suppuration, and after perforation of the pus gives rise to complete or incomplete rectal fistulæ. Cicatrices of the

intestinal mucous membrane sometimes give rise to stenosis and symptoms of ileus. A tendency to diarrhœa persists for life in some individuals. Dysentery may be associated with hepatic abscess, especially in tropical countries. The hepatic changes sometimes appear so early that we are inclined to believe that the dysentery and abscess of the liver are the result of the same agent. In other cases, the abscess is due to embolic processes in the mesenteric veins and portal vein. Gluck states that hepatic abscess is especially apt to develop after dysentery, if the liver is cirrhotic or waxy, as the result of previous malaria. Pylephlebitis has also been observed. Chronic Bright's disease is a rare complication, but cachectic œdema is occasionally observed. Burkart and Niemeyer found infarctions in the lungs and spleen, but only after the disease had lasted ten to fourteen days. Necrosis and diphtheria of the pharynx and larynx are rare. A fatal pyæmic or septicæmic condition occasionally develops, after having given rise to decubitus, pleurisy, pericarditis, parotitis, noma, or pseudo-erysipelas. Chronic dysentery may lead to waxy degeneration of the large abdominal glands. Multiple swellings of the joints, attended with pain and sweating, have been observed, sometimes followed by secondary disease of the heart. This is rarely noticed before the second week of the disease; it may result in ankylosis. From analogy with similar conditions in other infectious diseases, this must be regarded as a metastasis of the dysentery virus. Among the sequelæ must be mentioned paralyses, generally of a spinal character, and which were shown by Leyden to be the result of an ascending neuritis starting from the intestines. During violent diarrhœa, cramps sometimes appear in the calves or other group of muscles. The voice may become hoarse and high-pitched, as in cholera. Even the face may be sunken, the eyes surrounded by rings, and the skin clammy, while the internal temperature of the body is elevated. Signs of blood-dissolution (cutaneous hemorrhages, swollen gums) sometimes make their appearance.

III. Anatomical Changes.—The intestinal lesions of dysentery are generally confined to the large intestine, in rare cases they extend to the small intestines (usually the ileum). The lesions are most pronounced in the rectum, and gradually diminish upwards. The points of flexion of the large intestine are usually attacked with special intensity. This is attributed by Virchow to the fact that masses of fæces are apt to remain for a longer time in these localities, and thus exercise greater mechanical irritation upon the mucous membrane.

Upon opening the abdomen, the large intestine is generally found contracted and narrowed. Its serous layer is not infrequently very hyperæmic, and in places small subserous hemorrhages are visible. Its surface often looks opaque, and may be covered by a thin, veil-like membrane (beginning peritonitis).

The changes in the mucous membrane are sometimes catarrhal, sometimes diphtheritic.

In the catarrhal stage of dysentery, the mucous membrane of the large intestine is very congested, partly in a uniform manner, partly in the shape of individual dilated vessels. The congestion is most marked on all projecting parts of the mucous membrane, especially at the top of the folds and villi, and the three longitudinal columns of the colon. Subepithelial hemorrhages are found in many places, and may be merely punctate or cover a large surface. The mucous membrane is also swollen and its secretion increased; the surface is covered with an unusually

large amount of glassy mucus, often dotted or streaked with blood. The submucous tissue is also very succulent, congested, and swollen.

In the further course of the disease, the swelling of the mucous and submucous layers increases, while the hyperæmia diminishes. The secretion of the mucous membrane becomes opaque, and assumes a more purulent character. In some cases, the solitary lymph follicles take part in the swelling. They are enlarged, surrounded by a hyperæmic zone of vessels, and often undergo disintegration, at first in the centre, later at the periphery. The cavities of the follicles often contain mucoid, sago-like masses, such as are observed in the stools during life.

The microscope at first shows marked dilatation and congestion of the vessels of the mucous membrane and submucous tissue, with œdematous infiltration. The epithelium of the mucous membrane is intact, and the interstices between the individual Lieberkuehn glands appear broader than normal. At a later period, there is active emigration of white blood-globules, which accumulate in great numbers on the walls of the vessels. They are so densely aggregated in places between the glands of the mucous membrane, that the lower ends appear to be constricted. The more the œdema and emigration of white blood-globules increase the more the blood-vessels are narrowed and the hyperæmia diminishes. White blood-globules are also found on the walls of the vessels in the muscular coat

At first the enlargement of the follicles depends chiefly on congestion.and œdema, later upon hyperplasia of the cellular elements; if the latter becomes excessive, necrosis takes place.

If the dysenteric process advances, superficial epithelial necrosis and deposits are observed. The surface is covered with fine greenish-yellow or greenish patches, which cannot be readily removed. If forcibly removed, losses of substance are left over. In the most advanced cases, extensive necrosis occurs. The mucosa and especially the submucosa are very much thickened. The surface of the mucous membrane forms a nodular, greenish or blackish mass, the changes being especially marked along the longitudinal columns, and the projecting transverse folds. The microscope shows infiltration of the mucosa and submucosa with masses of fibrinous exudation. Destruction of the mucous membrane may give rise to dangerous hemorrhages, or gangrenous shreds hang into the lumen of the intestines, and deep-spreading suppuration of the submucosa and fistulæ develop. The extension of the inflammation may lead to peritonitis and perforation-peritonitis. Suppuration may also take place in the perirectal cellular tissue and terminate in rectal fistulæ.

After the acute inflammation has subsided, transverse ulcers of the mucous membrane are sometimes left over. These heal with difficulty, and give rise to chronic dysentery. Firm, callous cicatrices are a not infrequent sequel; they narrow the canal, and finally cause death from stenosis of the intestines.

The lymphatic glands of the mesocolon are generally swollen and congested; they sometimes undergo necrosis or caseation.

The other organs present no characteristic changes.

IV. Diagnosis.—A diagnosis is easily made, especially during an epidemic, from the character of the stools, the tenesmus, tormina, borborygmi, the tenderness on pressure and dulness in the left ileo-cæcal region. The disease may possibly be mistaken for: *a.* Rectal syphilis; syphilitic changes are found in other parts of the body. *b.* Rectal polypi; they are generally found in children and are recognized by digital examination. *c.* Hemorrhoids; the stools generally contain masses of pure blood; in addition, the dilated hemorrhoidal veins can generally be seen or felt.

d. Paramœcium, distomum, or anchylostomum; the ova of the parasites or the worms themselves are found in the stools.

V. PROGNOSIS.—The average mortality is seven to ten per cent, but in some epidemics it may reach sixty or seventy per cent. Cases of putrid and scorbutic dysentery have an unfavorable prognosis. Many patients die, after recovery from the dysentery, from marasmus or other complications.

VI. TREATMENT.—The prophylaxis is similar to that of typhoid fever.

The patients must be kept in bed and, apart from wine (especially astringent red wine), may take only soft-boiled eggs, meat broths, milk, and glutinous soups. A large cataplasm (not too heavy) is placed over the abdomen. So long as solid particles of fæces are found in the stools, mild laxatives (castor oil, calomel, etc.) should be given, and in mild cases these will produce recovery. In severe cases, they should be followed by an astringent. We prefer the following: ℞ Pulv. ipecac. comp., gr. v.; hydrarg. chlorid. mite, gr. ss.; sacch. alb., gr. v. One powder every three hours).

In addition, one to two litres ice-water, to which salicylate of soda has been added (two to four per cent), should be injected two or three times a day by means of Hegar's funnel. Carbolic acid should not be employed, inasmuch as poisoning with this remedy has been reported. Severe tenesmus may be treated with suppositories of opium or morphine; perhaps better still with cocaine.

Cures in Carlsbad, Marienbad, Kissingen, or Tarasp may be very useful in chronic dysentery.

Otherwise symptomatic treatment.

The following remedies have also been recommended: *a.* The various astringents (internally or by enema); *b.* narcotics: opium, strychnine, chloral hydrate, ergotin; *c.* laxatives; *d.* emetics; *e.* antiseptics (carbolic and salicylic acids, etc.); *f.* balsams: oil of turpentine; *g.* acids; *h.* potassium nitrate, potassium chlorate, etc.

5. *Asiatic Cholera.*

I. ETIOLOGY.—In 1830, Asiatic cholera first appeared in Europe. Originally endemic on the banks of the Ganges and Brahmaputra, it has several times left its habitat, and spread over almost the whole earth.

It is extremely probable, from Koch's celebrated investigations, that the cholera virus consists of the so-called comma bacilli. These are found constantly in the intestinal contents and walls.

They never appear in other organs or in the blood, urine, perspiration, lachrymal fluid, or inspired air. In one case, Nicati and Pietsch found them in the ductus choledochus, in five cases in the gall-bladder. Hence cholera seems to be the result of a local infection of the intestines.

The comma bacilli appear to be the virus of cholera, from the fact that they are constantly found in the disease, and also because inoculation in animals has produced cholera-like conditions. It almost seems as if the bacilli, in their growth, produce toxic substances. At least Nicati and Pietsch state that cholera stools and bacilli cultures emit, after a time, an ethereal odor, and that chemical substances are produced which cause symptoms of poisoning in dogs.

Comma bacilli are one-half to two-thirds as long as tubercle bacilli, but thicker and slightly curved. As a rule, the curve is not greater than that of a comma, more rarely it is almost semi-circular. The bacilli are sometimes in juxtaposition, and

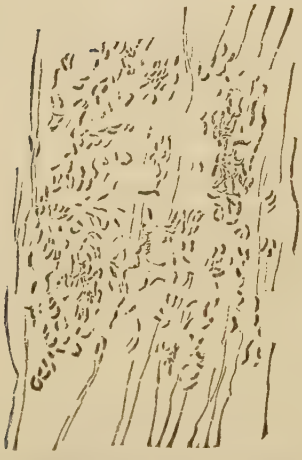
may then form S-shaped figures. They often grow into long threads, which are delicately convoluted like spirilli, so that Koch believes that they are not true bacilli, but constitute a transitional stage between spirilli and bacilli.

In dry preparations, they stain easily in a watery solution of fuchsin or methyl-blue.

In the moist condition, they proliferate very rapidly (vide Fig. 39), but soon die on drying. Cultures are readily made in meat infusion, peptone-gelatin, or on agar-agar, also in meat broth and milk. But the nutrient fluid should not be acid, since the bacilli are very sensitive to the majority of acids. Their proliferation ceases almost entirely below 16° C., but they remain alive even at 10° C. According to Raber, they die at 80° C. They also perish in media which contain numerous bacteria of putrefaction. Klebs suspects that the level of the barometer exercises an influence on their proliferation. Spores have not been discovered.

Curved bacilli are also found in diarrhœal stools, cholera morbus, tartar of the teeth, and cheese. Certain biological properties must also be present to permit the recognition of Koch's comma bacilli. Especially characteristic is their behavior in cultures in meat infusion and peptone-gelatin. At the start, the culture

FIG. 39.



Comma bacilli upon damp linen, from the dejections of cholera. Enlarged 600 times. After Koch.

FIG. 40.

Funnel-shaped retraction at the point of inoculation of the gelatin in the test tube. After Koch.

appears irregularly circumscribed, and has a granular appearance. Later the gelatin becomes fluid in the immediate vicinity of the colony, the latter sinks more and more in the gelatin, and finally forms a funnel-shaped depression, in the middle of which the colony is recognized as a small white spot (vide Fig. 40).

Cases have been reported, especially during the first epidemics, in which animals are said to have been attacked by cholera, but these statements cannot be regarded as reliable. The attempt has also been made to produce cholera in animals by feeding them with the vomit, stool, blood, urine of cholera patients, or injecting them into the blood-vessels or subcutaneously. Some experimenters obtained negative, others positive results; but it must be remembered that most of the substances employed were putrid, and that all the symptoms, accordingly, were probably the result of putrid infection. Koch found that the acid gastric juice of animals will destroy the comma bacilli.

Even human beings have made the disgusting experiments of swallowing the vomit and stool of cholera, and some suffered for their foolhardiness. That some escaped evil effects is not astonishing, if we bear in mind that the susceptibility to infection varies greatly in different individuals.

The virus of cholera is undoubtedly contained in the stools. In two cases Koch also found the bacilli in the vomited matters. The fæces are infectious in the fresh condition, and their virulent properties are intensified when they stagnate in a moist place. If they enter a water-closet, the germs are sometimes destroyed by excessive proliferation of putrefaction germs, but sometimes they meet with conditions favorable to their further development.

The risk of infection is run by every one who comes in contact with the stools (occasionally with the vomit). The germs first adhere to the body of the patient. Hence, cholera is a disease of human intercourse, and follows the paths of commerce and of armies. The more active the commercial intercourse between two countries the greater the danger that the disease will be conveyed from one to the other. During wars it has been found that the disease spreads along the track of the army, if this is suffering from cholera. In Asia, the disease has often been spread by pilgrims to various shrines.

Many cases are contagious, although the symptoms consist merely of slight diarrhœa. Such patients may spread the disease, and in this way an epidemic may apparently develop spontaneously.

Simple contact with a patient is not contagious, but the disease often attacks those engaged in cleaning the clothing or bodies of cholera patients. A house which contains the undisinfected stool of a cholera patient may become a source of infection for all its inhabitants. Hence, the rapid transfer of the patient to a hospital is not so important as the immediate removal of the healthy inmates of the house.

If the water-closets leak, their contents may contaminate the subsoil and drinking water. In East India, Koch found comma bacilli in a tank of water in which the clothing of a cholera patient had been washed. Nicati and Pietsch kept the comma bacilli alive for eighty-one days in the water of Marseilles harbor.

The germs of cholera may also be conveyed in the air, though this mode of infection is rare. This can result only from the mixture of infectious fluids with the air, inasmuch as the bacilli rapidly perish when in a dried condition.

Inanimate objects which are soiled with cholera stools or vomit are also capable of infection. Koch found unusually active proliferation of the bacilli upon wet articles of clothing.

Vegetables and fruit which are not thoroughly cleaned may also convey the germs. Koch stated that insects may act as carriers of comma bacilli, and Grossi found them upon the wings and belly of flies which alight upon cholera dejections.

In all probability, the infection is at first purely local from the gastro-intestinal tract. The general symptoms of cholera are probably the result in part of the enormous losses of water in the stools, and perhaps of the production of toxic substances by the bacilli.

It is assumed that the virus is generally inhaled into the naso-pharyngeal space, and then is swallowed with the food. It may also enter the stomach directly in infected drinking water or food.

Some individuals possess permanent or temporary immunity from the

disease. Sex exerts very little influence, although women are attacked somewhat more frequently. The majority of cases occur between the ages of 20 and 40 years, but the disease may also attack the new-born, children, and old people. In children, the disease is less frequent dur- the first five years than during the next five years of life. It is more frequent among the lower classes, chiefly on account of their poorer hygienic surroundings. It is said that negroes are especially apt to be attacked.

A predisposition may be created by excesses of all kinds, the administration of laxatives, or the ingestion of food which is apt to produce slight diarrhœa. A specially injurious influence is ascribed to mental excitement, particularly to the fear of contracting the disease. Cases have been reported in which the patients were attacked three times, and Stoufftet reports a case in which the individual experienced four attacks.

Cholera almost always occurs as an epidemic, or indeed as a pandemic which ravages the entire globe. The connection between the epidemics in different countries cannot always be readily traced, and they have been observed under very divergent external conditions.

Nevertheless, certain circumstances are known to act as auxiliary causes to the spread of the disease. In some towns the epidemic often starts from certain streets and houses, or rages with special violence in these localities. As a rule, those parts of a town suffer most which are situated on low, damp soil, and, moreover, those who live in cellars are attacked more frequently than others.

New, damp houses and overcrowded apartments offer favorable soil for the spread of the disease.

The majority of epidemics occur from June to August, the smallest number from January to March. High temperature, especially if protracted and followed by rains, favors the spread of the disease; cold weather and protracted rains oppose its extension. As a general thing, the more porous the soil, the more suitable it seems for the reception and proliferation of cholera germs (Pettenkofer).

In our opinion, this writer attaches too much importance to the level of the subsoil water. He claims that a high level is associated with increase, a low level with diminution of cholera. But good observers have shown that this does not always hold good.

Cholera sometimes attacks individuals who are suffering from other infectious diseases (measles, variola, pneumonia, intermittent fever, erysipelas, articular rheumatism).

The epidemics generally begin with a few imported cases. Many days, or even several weeks, often elapse between the introduction of the imported cases and the outbreak of the disease in others. The isolated cases rapidly multiply, and the epidemic reaches its height in four to six weeks.

At the beginning of the epidemic, the cases are generally more dangerous, and run a more rapid course than at the end. There are not infrequently numerous variations in the number of new cases, and in the mortality. The epidemic terminates gradually, and sometimes extends over many months.

If the cholera germs do not find a suitable soil, the single imported case, or a few cases of infection alone may be observed.

II. Symptoms.—The duration of the period of incubation is generally

estimated at two or three days. According to some writers, it occasionally lasts only two or three hours, in other cases four or five weeks (?).

We distinguish three different forms, according to the severity of the disease, viz.: cholera diarrhœa, cholèrine, and cholera asphyctica. The disease often begins in the mildest form, and gradually passes into the severest form. Transitional forms are also observed. In asphyctic cholera, and in cholerine (though not so frequently in the latter) recovery occurs gradually (stage of reaction). If, after the specific cholera symptoms have ceased, the patient is apathetic, and a condition develops resembling typhoid fever, we call the condition cholera typhoid.

It is not impossible that the cholera virus may produce milder symptoms than those of cholera diarrhœa. The psychical depression, palpitation, feeling of oppression, borborygmus, and cramps in the calves, which are observed in many individuals during cholera epidemics, have been attributed to cholera infection.

In many cases, cholera diarrhœa cannot be distinguished from diarrhœa due to ordinary causes. Its occurrence during an epidemic, and its infectious properties, especially the possibility that the fæces may give rise to severe cholera, are its chief characteristics. Its real nature is often doubtful, so that it is well to treat every case of diarrhœa, during the prevalence of an epidemic of cholera, as cholera diarrhœa. The discovery of the comma bacilli in the stools or successful cultivations will render the diagnosis positive.

In many patients, the symptoms appear during perfect health; in others, they are preceded by a cold, wetting, mental exeitement, error in diet, etc.

The patients not infrequently go to bed well, and are awakened from sleep by the first symptoms shortly before midnight or in the early morning. In others, the disease begins in the daytime.

The patients complain of rumbling in the belly, soon followed by a desire to go to stool. If they yield to this desire, they discharge at once an unusually large amount of fluid masses. These contain bile and, under the microscope, are also found to contain crystals of the triple phosphates, desquamated epithelium of the intestinal mucous membrane, débris of food, and innumerable round and rod-shaped schizomycetes. In the majority of cases, no pain is felt during the evacuation. Many become prostrated after the first discharge; in others, this is not felt until several stools have been passed. As a rule, five to ten evacuations occur daily, and sometimes they follow one another so rapidly that the patients, at times, do not dare to leave the bed-pan. The more profuse the diarrhœa the less the excretion of urine. Finally, only a few drops of urine are voided after straining and severe burning in the posterior part of the urethra. Albumin is almost always present in the urine.

Some patients complain at the start or soon afterwards of nausea, singultus, and even vomiting. The mouth is sticky and hot, the tongue is thickly coated, there is often fœtor ex ore. Thirst is increased, while the appetite is lost. The patients not infrequently experience a sensation of oppression and extinction, complain of palpitation and spasms in the cardiac region, the skin becomes cold and pale, the face pinched, the pulse small, and there may be cramp-like pains in the muscles, especially the calves.

If aid is rapidly brought, the disease may terminate in a few hours. In other cases, it lasts several days to a week and then terminates spontaneously, or it passes into the severe forms of cholera. Children, old

and exhausted individuals sometimes die from cholera diarrhœa alone, with symptoms of increasing collapse.

But the symptoms are not infrequently so mild that the patients go about their business and even travel long distances, depositing foci of infection wherever they have an evacuation from the bowels. Even the mildest cases, however, may suddenly become severe.

The symptoms of cholerine often develop from those of cholera diarrhœa. The numerous fluid stools likewise constitute the chief symptom in this form. But the more frequent and copious the evacuations the more they lose their biliary character and finally form a colorless or gray fluid, mixed with yellow or gray flakes (rice-water stools). At the same time the fæcal odor is lost, and the stool acquires a sickish odor. The urinary secretion diminishes and ceases earlier than in cholera diarrhœa. Vomiting is a constant symptom, at first of the contents of the stomach, later of rice-water masses, similar to those discharged per rectum. The other symptoms are similar to, but more severe than, those of cholera diarrhœa.

The symptoms may cease within twenty-four hours, after ten or fifteen evacuations. Recovery sometimes takes place with surprising rapidity, at other times it is slow and attended with symptoms which remind us of the stage of reaction of asphyctic cholera. So long as the stools contain bile, the prognosis is not very grave. But if rice-water stools appear and persist for some time, asphyctic cholera often develops, and a fatal termination is then the rule, rather than the exception.

In asphyctic cholera, the chief symptoms are the rice-water stools and vomit. The other symptoms are in great part the result of the great losses of water. Hence the blood is thickened, and thus gives rise to a series of circulatory disturbances (imperceptible pulse, deep cyanosis, associated with pallor, cold skin, loss of turgor in the various tissues, anuria, painful muscular cramps, etc.).

The number of evacuations varies; there may be more than twenty or thirty in the twenty-four hours. The diarrhœa is often especially frequent in the beginning of the disease. Cessation of diarrhœa is not always a favorable sign, since this is observed in very feeble individuals shortly before death, when the muscular coat of the intestines is paralyzed. At the autopsy, the intestines are found filled, in such cases, with large amounts of fluid. Involuntary evacuations are also observed in patients who are in a condition of collapse.

The amount of the stools is often larger than that of the food ingested, positive evidence that the excess is derived from the blood. The daily amount may vary from 500 to 5,000 ccm.

The reaction of the rice-water stools is usually alkaline or neutral; their specific gravity varies from 1.006 to 1.013. They have a gray color and contain the characteristic flocculi. The stools often assume a reddish color on the addition of nitric acid.

Microscopical examination of the cholera stools reveals detritus of food, a few triple phosphates, a few round cells, still fewer red blood-globules, round and rod-shaped schizomycetes, punctate masses and drops of fat. Intestinal epithelium cells are often almost entirely absent, in other cases we find a few shreds, often coherent cells. The latter form part of the flocculi. The latter contain nothing else beyond a punctate mass, and are then composed of mucin and a trace of albumin.

The comma bacilli are obtained by spreading one of the flocculi upon

a cover glass, drying it by drawing it several times through the flame of a spirit lamp, and then covering it with an aqueous solution of fuchsin or methyl blue. The cover glass is then rapidly washed in water, again dried, and imbedded in Canada balsam (vide Fig. 41).

Despite every precaution, no bacilli may be found, and it then becomes necessary to make cultures with the flakes of mucus.

The rice-water stools contain a large percentage of water, and a small amount of solid constituents, especially of organic matters. Albumin is present, if at all, in mere traces. Urea and carbonate of ammonia are also present. Sodium chloride is the chief inorganic constituent, the phosphates and potassium salts are present in very small amounts. Kuehne found a sugar-producing ferment in all cases, and this led Cohnheim to assume that the stools are not a simple transudation from the vessels, but intestinal secretion. According to this theory, the virus of cholera gives rise to hypersecretion of the glands of the intestinal mucous membrane. The excessive secretion stimulates peristalsis and thus causes frequent evacuations; this probably gives rise, by reflex action, to increased excitability of the muscular coat of the stomach and thus causes the frequent vomiting.

Fig. 41.

Comma bacilli from a flake. After Koch.

The older writers applied the term cholera sicca to cases in which, while the patients had other choleriform symptoms, they rarely or never had an evacuation from the bowels, but the autopsy showed that the intestines contained a large amount of fluid. In an epidemic in Kœnigsberg, in which I treated nearly one hundred cases of cholera, I observed one case of this kind. In the Genoa epidemic of 1883, a comparatively large number of such cases appear to have been observed.

Vomiting occurs in almost every case of asphyctic cholera, sometimes more than twenty times a day. Emesis is generally very easy, and the fluid often bursts in streams from the mouth. As a general thing, it is so much more profuse the more the patients yield to the feeling of thirst. When the patients are prostrated, the vomiting may be replaced by singultus which often produces violent epigastric pains. Singultus and vomiting sometimes alternate with one another.

The vomited masses consist at first of débris of food, later they are stained with bile, and then present the characteristics of the rice-water stools. They may amount to 35,000 ccm. in twenty-four hours. Goldbaum calculated that one of his patients drank 10,200 ccm. of fluid and vomited 21,250 ccm. The amount discharged with each act of vomiting varies from 30 to 500 ccm.

It is evident that some of the fluid is derived from the blood and probably enters the stomach from the intestinal canal. Its reaction is

usually alkaline or neutral, its specific gravity varies from 1.002–1.005, and contains only a small amount of solid matter.

The microscope generally shows drops of fat, even if fat has not been ingested, epithelium cells from the œsophagus and buccal cavity, a few round cells, and various schizomycetes, rarely comma bacilli.

The abdomen, as a rule, is slightly sunken; the lower part is more prominent than the upper. There is generally no tenderness on pressure. Below the umbilicus we often feel a peculiar shaky feeling of resistance and gurgling, and find dulness on percussion.

The appetite is lost, but the patients are tortured by thirst. If too much is drunk, vomiting is produced. The majority of patients complain of burning in the mouth and internal heat, especially in the gastric region. The tongue may be clean and unusually red, or it is covered with a gray coating; it is usually dry and sticky.

The loss of water, as a matter of course, gives rise to gradual drying of the various tissues. Not alone is the amount of blood diminished, but it becomes thicker and offers greater resistance to the propelling power of the heart. The friction in the peripheral vessels also increases. The circulation becomes slower, the blood cools at the periphery of the body, and this again slows the circulation. At the same time the nutrition of the tissues suffers.

The skin is extremely pale and, at the same time, intensely cyanotic, particularly the lips, cheeks, tip of the nose, conjunctiva, and finger nails. The tongue also is often cyanotic.

The features are extremely sunken. The malar bones and nose are prominent, the eyeballs are deeply sunken and surrounded by bluish gray rings. This, together with the diminished power of the muscles, interefres with closure of the lids. At first the patients can close the lids when so directed, but they soon open again. But in a little while they lie constantly with half-closed lids, and the eyeball rolled up until the cornea is entirely covered by the upper lid. According to Graefe, the rolling upwards of the eye is only apparent, and produced by the imperfect closure of the lids. The patient acquires such a peculiar expression as to justify the use of the term facies cholerica.

The exposed portions of the conjunctiva are often dry and devoid of gloss. Along the lower edge of the cornea we not infrequently find intense congestion of the subconjunctival vessels. Joseph observed subconjunctival hemorrhages in some cases. The absorption of substances which are dropped into the conjunctival sac takes place very slowly. Joseph states that the patients are unable to weep.

The sclera sometimes contains irregular bluish and blackish patches, arranged concentrically around the lower edge of the cornea (partial desiccation and thinning of the sclera). They are said to occur only in fatal cases.

The lowermost portions of the cornea sometimes contain dry brownish patches, which leave opacities if recovery takes place. In unfavorable cases, softening and destruction may occur.

The pupils are usually contracted, perhaps from paralysis of the sympathetic.

Choroidal hemorrhages have been observed in some cases.

The ophthalmoscope shows that the retinal arteries are unusually narrow, but intensely red. Slight pressure on the globe suffices to produce pulsation in the retinal arteries, and if the pressure is increased, complete anæmia develops. The retinal veins are very wide and bluish-red in color ; in places they are sometimes empty. The optic disk often has a pale lilac color, its centre is somewhat paler.

Vision is generally unaffected. The obscuration of the field of vision, which

is observed particularly at the beginning of the disease, is probably the result of purely nervous disturbances.

The skin, particularly on the forearms and backs of the hands, is wrinkled and flabby. If it is cut, the edges of the wound do not gape; if they are separated, they do not exhibit any tendency to approach one another. Vesicles can no longer be produced by the application of blisters or the actual cautery. Not infrequently the skin feels clammy, moist, and ice-cold (cholera algida).

The panniculus adiposus is flabby and diminished in size. The absorptive properties of the subcutaneous tissue are impaired, but not abolished.

For example, Goldbaum produced dilatation of the pupils by subcutaneous injection of atropine. Sodium salts, which were injected subcutaneously, were found in the evacuations, potassium salts were found in the saliva, but not in the stools.

Painful muscular spasms are an extremely annoying symptom of cholera. They are most frequent in the calves, next in the thighs, forearms, and fingers, rare in the muscles of the abdomen and chest, most rare in the face. When they occur in the calves, the thighs and legs are involuntarily flexed spasmodically, and in lean individuals the hard muscles can be readily seen and felt through the skin. The pains are so severe that many patients cry out aloud, and the spasms may recur at intervals of less than ten minutes. The more profuse the stools and vomiting the more violent are the cramps, though the latter sometimes precede the former. They are supposed to be due to dryness of the muscular substance, but, in our opinion, nervous causes cannot be excluded.

Some authors mention increased mechanical and electrical excitability of the muscles. Josias has found that, in severe cases, the tendon reflexes were increased at the height of the disease, and became normal during convalescence.

Consciousness is generally retained to the last moment; in rare cases, delirium occurs towards the end of life. The majority of patients manifest, at a very early period, a sort of fatalistic mood and feeling of indifference. Many complain of precordial terror, dizziness and ringing in the ears, and suffer from syncopal attacks.

The weight of the body diminishes very rapidly, chiefly on account of the abundance of the evacuations from the bowels. If the disease runs a very rapid course, there may be a loss of one-half to one per cent of the weight of the body within an hour.

The bodily temperature always appears subnormal to the hand, and the thermometer in the axilla also, as a rule, shows subnormal temperature. The thermometer rises so slowly that it should be left at least half an hour in the axilla. The mouth and tongue generally feel cool. The vaginal and rectal temperature, however, is rarely subnormal, somewhat more frequently normal, and, as a rule, increased to 40° C. (there may be a difference of 3.7° C. between the rectal and axillary temperatures). Shortly before death, the temperature sometimes increases rapidly to 42.4° C.

The pulse is accelerated and not infrequently irregular. The more profuse the vomiting and evacuations the more it diminishes in ful-

ness, until finally it becomes imperceptible. The pulse disappears earliest in the radial artery, later in the more central arteries, latest in the carotids.

The blood is generally of a deep black-red color, tarry, and grows red to only a slight extent or not at all when shaken in the air. It is often acid before death.

The number of white blood-globules is generally increased, and they are often collected in masses. On account of the concentration of the blood, the red blood-globules are unusually close together, and exhibit but little tendency to form rouleaux. In a few cases, free drops of fat have been found in the blood.

The specific gravity varies from 1.036–1.058 (normally 1.026–1.029). The amount of albuminoids and potassium salts in the blood is unchanged, that of sodium chloride is diminished.

C. Schmidt furnishes the following analyses:

	Healthy woman, æt. 30 years.	Woman, æt. 26 years, 36 hours after the beginning of an attack of cholera.
Water	824.55	760.85
Solid matters	175.45	239.15
Hæmoglobin	116.43	154.30
Fibrin	1.91	3.50
Other organic substances	48.49	74.35
Inorganic salts	8.62	7.00
Sodium chloride	2.845	1.953

If the arteries are laid bare, they appear narrow and transparent. Upon opening them, a thin stream of dark blood escapes, and in severe cases none escapes, unless the vessel is stroked towards the periphery. This manipulation is then followed by the escape of a few thick drops of blood. Dieffenbach found in one case that no blood followed after opening an artery in the arm, although he introduced a catheter into the vessel for a considerable distance.

The veins are distended with blood, but venesection is not followed by a vigorous stream. On the other hand, it can often be removed only by stroking the limb.

On account of the impaired nutrition, there is diminished sensibility of the mucous membranes. This is noticeable upon the conjunctiva and the mucous membrane of the nose and air passages.

The voice becomes muffled and peculiarly high (vox cholerica). This is the result of dryness of the larynx and weakness of its muscles. Matterstock found that the processus vocales were unusually prominent, and that the glottis opened widely in attempts at phonation. These symptoms were sometimes found only on the left side.

The respirations are not infrequently accelerated, deep and irregular, owing to the disturbances in the pulmonary circulation. The expiratory current of air is often unusually cold.

The heart's action is not infrequently accelerated and irregular; it grows feebler the greater the general prostration. The first sound becomes indistinct, and the diastolic sounds disappear entirely. In a few cases, pericardial friction sounds are heard. These are generally attributed to excessive dryness of the pericardium, but result in some cases, perhaps, from subepicardial hemorrhages.

The excretion of urine generally ceases entirely in a short time; if small amounts continue to be passed, they generally contain albumin.

The complications of asphyctic cholera generally appear during the stage of reaction. We may mention bloody stools (usually an unfavora-

ble sign) and hæmatemesis. The latter is commonly small in amount and produced by violent vomiting.

Cutaneous emphysema is a rare complication which is secondary to interstitial pulmonary emphysema, produced by the violent respiratory movements.

The majority of cases of fully developed asphyctic cholera terminate fatally. Death may occur before the end of the first day, or life may be prolonged for forty-eight to seventy-two hours. Life gradually becomes extinct from excessive weakness of the heart and general prostration. The danger is not always ended when the symptoms improve, inasmuch as relapses are apt to occur, and severe sequelæ develop in not a few cases.

The period between the cholera attack proper and pronounced recovery is known as the stage of reaction.

It is not often that this runs its course without serious disturbances. The stools then become less frequent, firmer, and stained with bile; vomiting ceases, diuresis is re-established, and the temperature, pulse, and respirations again become normal. But the patients must keep a strict diet for a long time, in order to prevent grave relapses.

In a second group of cases, the reaction is accompanied by slight fever and congestive symptoms. The conjunctivæ are injected, the face flushed, many patients complain of rush of blood to the head, and delirium sometimes sets in. In a few days, these symptoms subside and recovery becomes complete.

In a third series of cases, the condition, in its gross features, resembles typhoid fever (cholera typhoid). The temperature rises considerably, the pulse is accelerated, full and tense, the tongue is dry and covered with sordes, the abdomen is tympanitic, and not infrequently roseolar patches appear on the trunk; in addition, diarrhœa and clouded consciousness.

Grave dangers arise in the stage of reaction if the renal secretion is not re-established and uræmic symptoms appear. This will occur if the renal circulation has been interrupted, or almost interrupted, for such a long time that the endothelium of the Malpighian bodies and the tubular epithelium are rendered incapable of function. In some cases, no more urine is secreted; in other cases, the urine is abnormal. Hence, urea accumulates in the blood and tissues. Crystals of urea have even been observed upon the skin (urhidrosis). The uræmic form of the stage of reaction not infrequently presents a typhoid character, although not all cases of cholera typhoid are uræmic in character.

The anuria occurring in cholera is the result of the low blood pressure and the slowness of the circulation. It may continue after the attack of cholera, and sometimes lasts six days. The longer its duration the more certain is the fatal termination. According to Goldbaum, recovery never takes place if anuria persists for more than seventy-two hours.

The quantity of urine passed on the first day after an attack of cholera varies from 30 to 500 ccm. (about 200 ccm. on the average). It gradually increases and may terminate finally in polyuria. The amount does not become normal until the second week. The urine generally has a reddish or reddish-brown color, and is ordinarily cloudy. Its reaction is almost always acid; sometimes so intensely that Stokvis suspects the presence of an unknown free acid. In one case, this observer found a neutral reaction. The specific gravity varies from 1009 to 1025.5 (1015

on the average). The sediment contains round cells, a few red blood-globules, often very numerous and, in part, fatty epithelium cells from the renal tubules, vesical epithelium, and casts. The latter are partly hyaline, partly granular, and are covered with drops of fat and epithelium cells. Their diameter varies, and they are often unusually long. According to Wyss, the prognosis is so much more favorable the more numerous the casts, because in this way the tubules are rendered pervious. The sediment often contains crystals of uric acid. Nedwetzky found spermatozoa.

The urine which is first voided almost always contains albumin. In the subsequent portions the albumin diminishes, and disappears before the amount of urine has returned to the normal. Stokvis noticed that the higher the specific gravity of the urine the smaller the amount of albumin. It generally disappears between the fifth and eighth days, but Wyss has found it as late as the thirteenth day.

Often, though not constantly, the urine contains a substance which reduces an alkaline solution of copper sulphate. This is generally believed to be sugar, but Wyss thinks that it may be glucose, and be produced by decomposition of the indican which is present in large quantities in cholera urine. Glycosuria may occur after, or coincidently with, albuminuria. In some cases, Wyss found that the glycosuria was most marked from the fifth to eighth days, and lasted a week.

The amount of urea is very slight (2.5% on the average) in the first cholera urine. If recovery occurs, it gradually increases and may become excessive by the beginning of convalescence (two and a half to two and three-quarter ounces). Stokvis thinks that the amount of kreatinin is increased. Only a trace of sodium chloride is present, but the quantity gradually increases. According to Panchet, the urine contains an abundance of the salts of the biliary acids. The urine also contains a large amount of indican.

The stage of reaction may be associated with a large number of complications, which sometimes begin during the cholera attack proper.

Various exanthemata are observed upon the skin. Herpes labialis is one of the rarer symptoms. Eruptions resembling urticaria, scarlatina, or measles occur frequently, and are sometimes followed by desquamation. Scattered roseolar patches appear occasionally. In some cases, pemphigoid or impetiginous eruptions are observed. Multiple cutaneous abscesses sometimes develop and may last for weeks after the attack of cholera. Miliaria and erysipelas have also been described.

Gangrene of the skin or certain parts of the extremities develops in some cases. It sometimes follows wounds of the skin, for example, after blistering or leeching. In some cases, cutaneous changes are followed by thrombosis of the vessels. Gangrene of parts of the limbs has also been described, as the result of arterial embolism, which has taken its origin in cardiac thrombi or in recent endocarditic deposits. Marantic thrombosis of the veins may also occur.

The skin is sometimes more or less anæsthetic or hyperæsthetic. Muscular contractures, of central or peripheral origin, have also been observed. In a few cases, the joints undergo painful swelling.

Some authors describe violent delirium during convalescence, and even maniacal attacks occasionally develop. On the other hand, improvement and even complete recovery of previous psychopathies have been reported in other cases of cholera. Among other nervous compli-

cations may be mentioned: general convulsions, trismus, tonic and clonic spasms, monoplegia, paraplegia, and hemiplegia, rarely meningitis, and chorea (in children).

Catarrhal or diphtheritic changes are observed on the buccal mucous membrane, and salivation has been reported in a few cases. Purulent parotitis (more rarely inflammation of the submaxillary gland) has also been mentioned, and, according to Gueterbock, is always secondary to catarrh of the excretory ducts of the glands. The lesion is sometimes bilateral, and may terminate fatally on account of burrowing of pus, erosion of vessels, or œdema of the glottis. Paralysis of the tongue and palate has been observed in one case.

Diphtheritic changes may also develop upon the mucous membrane of the pharynx, œsophagus and stomach, duodenum and large intestine (tenesmus and bloody stools).

Bronchitis is not uncommon; less frequently there is necrosis or œdema of the laryngeal tissues. Pneumonia, abscess, hemorrhagic infarctions, gangrene of the lungs, and pleurisy are observed occasionally.

Icterus and peritonitis are rare complications. Women often suffer from pseudo-menstrual bloody discharges. Gangrene of the genitals may develop in both sexes.

Pregnancy does not protect against cholera. When pregnant women are attacked, premature delivery is often produced. The fœtus sometimes dies in utero; in other cases, it is born alive, but is attacked by cholera immediately after birth, or a short time afterwards.

In nursing women, the lacteal secretion is not affected by an attack of cholera, and the milk does not appear to be infectious.

The sequelæ include chronic Bright's disease and diabetes mellitus. In some instances, it is said that diabetics recovered temporarily or even permanently after an attack of cholera.

III. Anatomical Changes.—When death occurs during the cholera attack proper, the corpse often presents a peculiar appearance.

The body is pale, but deep cyanosis is exhibited in the lips, tip of the nose, and finger nails. The features are sunken and peaked. Rigor mortis is unusually pronounced. The arms, legs, and fingers are flexed, and the contours of the muscles are distinctly visible under the skin. The corpse cools very slowly, and there is sometimes a post-mortem rise of temperature. Decomposition sets in very slowly.

Post-mortem twitchings of the muscles constitute a noticeable feature. They generally begin immediately after death, sometimes not until fifteen minutes later. They appear first in the lower limbs, and then extend to the arms, trunk, and face. The calves are usually unaffected. The twitchings are so much more active the more rapid and violent the course of the disease has been. In many cases, they are capable of changing the position of the body, and they may continue for two and a half hours. Drasche states that the skin over the contracting muscles is reddened uniformly or in patches, and that its temperature may rise 0.7° C. Contractions could be brought about by pouring chloroform on the skin and allowing it to evaporate. The cause of this phenomenon is unknown.

Similar phenomena appear to occur in the involuntary muscles. Drasche observed cutis anserina above the contracting muscles, and states that in one case a discharge of semen took place one and a half hours after death.

The subcutaneous cellular tissue and muscles are generally very dry, and the muscles are unusually dark and red. The medulla of the bones is also extremely red.

The serous membranes have a sticky, soapy feel, and their cavities do not contain the ordinary post-mortem transudation.

More or less extensive hemorrhages are found not infrequently beneath the epicardium and also in the heart muscle. The right heart and main venous trunks are filled with blood, the left heart is empty. Fatty and waxy degeneration is observed in the muscular fibres of the heart.

The blood has a tarry color and consistence, and is very slightly or not at all coagulated.

The lungs are strongly retracted and pale; on section, a few thick drops of blood can be squeezed out of the larger vessels. The consistence of the lungs is leathery and tough.

The loops of the small intestine are filled with rice-water masses, and agonal invaginations are often present. The serous layer is injected and often cyanotic. The mucous membrane is swollen. The top of the folds and villi is often congested, and sometimes infiltrated with blood. The solitary follicles and Peyer's patches are swollen and may be surrounded by a halo of distended vessels. In the beginning of the disease, the swollen follicles, if punctured, discharge a clear fluid and then collapse, but later the swelling is produced by hyperplasia of the lymph cells and is not diminished by puncture of the follicles.

The intestinal epithelium is elevated in places by serous fluid, or is exfoliated in more or less extensive shreds. This may occur during life, but is also, in part, a post-mortem change. These changes are most marked in the lower part of the ileum, but in very rapid cases have been traced to the pylorus.

Kelsch and Renault found marked infiltration of round cells in the subepithelial and subserous connective tissue, considerable dilatation of the submucous vessels, and occlusion of the lymphatic vessels, partly with round cells, partly with desquamated and swollen epithelium. Lieberkuehn's glands were dilated in part with mucus, and the epithelium of the upper portions often absent (probably a post-mortem change). Koch found comma bacilli in these glands, partly in their lumen, partly between the epithelium and basement membrane. Other bacteria are also found, but appear to have entered after the death of the tissues.

In some instances, especially in very rapid cases, the intestinal contents form almost a pure culture of comma bacilli.

Small losses of substance are sometimes produced in the central portions of the swollen lymph follicles by hemorrhagic infiltration and softening.

The large intestine may appear intact. In other cases, there is congestion of the serous and mucous membranes, with swelling of the latter. The microscopical changes are similar to those found in the small intestines. This is also true of the stomach.

Goldbaum describes swelling of the circumvallate papillæ of the tongue as a constant appearance.

The mesenteric glands are often congested and slightly swollen.

The spleen may be slightly enlarged.

The liver is slightly anæmic, the gall-bladder occasionally distended with watery bile or with a serous fluid. According to Nicati and Rietsch, the mouth of the ductus choledochus is often occluded by epithelial and mucous masses; the functions of the liver are soon abolished, and its weight diminishes considerably. They believe that acholia plays a part

in producing the fatal termination. In one case, Goldbaum described diphtheria of the gall-bladder.

The kidneys are pale, but hyperæmic in places. The interstitial tissue is œdematous. The tubules contain numerous red blood-globules. The epithelium cells of the tubules are partly granular, partly desquamated, and in places in a condition of coagulation necrosis. These changes are degenerative in character, and result from the imperfect circulation of the blood.

Hemorrhages and exfoliation of epithelium are often observed upon the mucous membrane of the renal pelvis and the bladder. The latter is generally empty or contains a few drops of urine mixed with mucus and shreds of epithelium. Diphtheria of the vesical mucous membrane is sometimes observed.

The mucous membrane of the uterus is not infrequently swollen and suffused with blood, and may also contain blood on its free surface. Hemorrhages are sometimes found in the ovaries.

The sinuses of the dura mater are usually filled with blackish blood. The pia mater is often moist and sticky. Meningeal and cerebral hemorrhages have been described.

IV. Diagnosis.—During an epidemic of cholera, the disease is easily recognized. This is not true of the first cases, in which chief reliance must be placed on the discovery of comma bacilli. In distinguishing Asiatic cholera from cholera morbus, it must be kept in mind that the latter may occur in our latitudes at any time, and that it rarely terminates fatally. According to Finkler and Prior, the stools of cholera morbus also contain curved bacilli which look very much like Koch's comma bacilli. Whether the former are found constantly in cholera morbus is still undecided.

The bacilli of cholera morbus are thicker and less curved than the comma bacilli, they have more of a spindle shape with rounded ends; they develop fewer threads which are not so long or so twisted as those of the comma bacilli. Cultures of Finkler's bacilli on plates of nutrient gelatin first form regular disks with smooth borders, which liquefy the gelatin much more vigorously than do the comma bacilli. The two varieties of bacilli also present other biological differences.

The diarrhœa of cholera presents no specific characteristics, so that it is well to regard as choleraic every diarrhœa which occurs during an epidemic. Poisoning with arsenic, tartar emetic, and corrosive sublimate, and incarceration of the intestines may be mistaken for cholera during the prevalence of an epidemic. Even the gross anatomical lesions of arsenic poisoning may be similar to those of cholera, so that crimes may escape discovery during an epidemic. The diagnosis is rendered positive by the discovery of comma bacilli in the intestinal contents.

V. Prognosis.—As a matter of course, the prognosis is so much more favorable the milder the form of the disease. In asphyctic cholera recovery is exceptional. The average mortality is about sixty per cent of all cases, although it varies greatly in different epidemics. The sudden cessation of vomiting and diarrhœa is regarded by many as an unfavorable sign, and the formation of blackish specks on the sclera is considered a forerunner of impending death. After the symptoms of cholera have subsided, the prospects of recovery are so much more favorable the

earlier the urinary secretion is re-established. If this does not take place within three days, death is almost inevitable.

VI. TREATMENT.—A country can be protected against the importation of cholera from an infected country only by the strictest quarantine, although practically this is extremely difficult to secure. Those vessels which come from tropical cholera districts must be watched with special care.

When cholera appears in any locality, the inhabitants must be warned against excesses of all kinds, against eating unripe food or vegetables, or anything which is liable to produce diarrhœa. Suspicious wells or other sources of water supply must be closed, and it is best to drink only water which has been boiled and to which brandy or wine has been added. Festivals and other large gatherings should be prohibited. If diarrhœa sets in, medical aid should be sought at once. Water-closets are to be disinfected daily.

If cholera appears in a certain house, the healthy inhabitants should be removed forthwith. Special care must be devoted to the disinfection of the stools, vomited matter, and clothing. The best disinfectants are carbolic acid (5%) or corrosive sublimate (1 : 1000).

Cholera corpses should be placed in hermetically closed caskets and should be buried privately and quickly.

A patient suffering from cholera diarrhœa should be kept in bed, and given only red wine and meat broth (mutton). We may order the following prescription:

℞. Tinct. valerian. æther.,
Tinct. opii simp. ..... āā 3 ss.
M. D. S. Ten to twenty drops every three hours.

Italian physicians recommend intestinal infusions of a lukewarm solution (one per cent) of tannin (one liter).

Cholerine may be treated in a similar manner. The violent thirst is quenched with pieces of ice, severe vomiting by subcutaneous injections of morphine in the epigastrium, cramps in the calves by injections of morphine in this region. A warm poultice should be applied to the abdomen. In asphyctic cholera, warming flasks are placed in the bed, and a warm poultice on the abdomen; wine, brandy, and champagne are given as stimulants, ice to relieve thirst; internally, we may order laudanum or

℞. Pulv. ipecac. comp. ..... gr. vij.
Hydrarg. chloride mite. ..... gr. iss.
Sacch. alb. ..... gr. vij.
M. To be taken every three hours.

Cramps in the calves may also be treated by friction with dry cloths, alcoholic inunctions, or mustard poultices.

In the stage of reaction, extensive use should be made of lukewarm baths (28° R. twenty to thirty minutes' duration, t. i. d.); otherwise symptomatic treatment. Great caution must be exercised for a long time as regards diet.

The following are some of the other remedies which have been employed in this disease: *a*. narcotics: opiates, strychnine, calabar, curare, belladonna, ergotin, chloroform, chloral hydrate, amyl nitrite, etc.; *b*, styptics: tannin, bismuth, alum, etc.; *c*, drastics; *d*, emetics; *e*, nervines: arsenic, nitrate of silver,

etc.; *f*, antiparasitics: carbolic acid, calomel, quinine; *g*, venesection; *h*, transfusion of blood, sodium chloride solution, or milk; *i*, subcutaneous infusion of sodium chloride solution in large amounts; *k*, enemata of starch, narcotics, and styptics; *l*, inhalations of oxygen.

6. *Yellow Fever.*

I. Etiology.—The home of yellow fever is the West Indies, particularly the greater Antilles. It is also endemic on the gulf coast of Mexico and on the west coast of Africa.

From these regions the disease has often been conveyed to other parts of America, and also to European countries, especially to sea-ports.

The spread of the disease is associated with communication by water, and hence it appears chiefly in sea-ports or cities situated on large rivers. It often appears on shipboard; or vessels coming from yellow-fever localities, despite the fact that the disease does not appear upon the vessels themselves, infect other ports when they discharge their cargo, bilge water, or other refuse.

It was formerly supposed that the disease may develop autochthonously upon vessels as the result of bad ventilation, overcrowding, and stagnant bilge water, but these conditions simply offer a favorable soil for the virus.

The nature of the virus is unknown, but it probably consists of bacteria. The virus directs its attack mainly against the liver, and gives rise to changes similar to those of acute yellow atrophy of the liver.

The outbreak of epidemics depends mainly on the temperature. The majority of American epidemics appeared from July to September. They generally disappear rapidly after the occurrence of frost. The continuance of the epidemic is favored by protracted rains or great moisture of the atmosphere.

Those parts of the city are in greatest danger which are nearest to the harbor or banks of a river. The poorer the hygienic conditions in any quarter of the town the more favorable the conditions for the spread of the disease.

Negroes escape almost entirely. In American epidemics, recent immigrants are most liable to be attacked, and the danger lessens the longer they are acclimated. If they leave America for a time and then return, the susceptibility to the disease also returns. According to recent reports, the importance of acclimatization has been greatly overestimated.

The male sex is more affected, especially in middle age. Old people and infants are rarely attacked. Jones reports a (doubtful) case of infection of the fœtus by the mother. It is said that those who are much exposed to heat (bakers, cooks, etc.) are very susceptible, and that those who are accustomed to foul odors (tanners, soap-makers) are not attacked. Excesses of all kinds favor the outbreak of the disease.

More than one attack is very rarely experienced by the same individuals, but relapses are more frequent.

The infection is not conveyed from one individual to another by simple contact, and the virus does not appear to proliferate in the body of the patient. He simply acts, like inanimate objects, as a carrier of the virus.

II. Symptoms.—The average duration of the period of incubation is said to be two or three days. Some authors claim to have observed an incubation period of only a few hours, others of two weeks or even more.

The disease is often preceded by prodromata (nausea, anorexia, malaise, etc.).

The disease begins not infrequently with a single vigorous chill or several slighter chills. The bodily temperature rises very rapidly and soon reaches 40° C. or more. At the same time, the pulse is accelerated (one hundred to one hundred and twenty beats or more). The patients complain of throbbing in the head, and intolerable unilateral or bilateral pains in the temples, more rarely in the occiput or other parts of the body. The gaze is fixed, the conjunctiva injected. The tongue has a gray or grayish-yellow coating. There is nausea and not infrequently repeated vomiting. The patients feel unusually weak and despondent.

They often emit a cadaverous odor, which Stoone claims to have detected before the outbreak of other symptoms. It is said that the patients, after recovery, are no longer bitten by mosquitoes. On the other hand, it is claimed that the disease may be conveyed by these insects. The majority of patients complain of severe pains in the loins, and pains in the joints and muscles are also common.

The heart and lungs remain unaffected.

The gums are often loose, and covered with desquamated epithelium;

this may be followed by ulcers and hemorrhages. The epigastrium is often sensitive. There is usually constipation, rarely diarrhœa. The urine grows scanty and soon contains albumin. Complete anuria occasionally develops.

This stage of the disease lasts one to four days. It is followed by the second stage (stage of remission), which generally lasts one or two days. Profuse diaphoresis suddenly appears and the temperature and pulse become normal in a few hours. The patients feel better, and sometimes recovery ensues forthwith. Next follows the stage of blood dissolution and jaundice, which lasts, on the average, one to three days. An icteric color of the conjunctivæ has not infrequently been noticed on the preceding days. This now increases and extends to the general integument, and may attain the most intense grade possible.

Diuresis, which had increased during the period of remission, again becomes scanty, and the urine contains a large amount of bile pigment, while the biliary acids are sometimes absent. Anuria and death from uræmia are not infrequent. Ullersperger described lipuria.

This author found that the blood was dark, some of the red blood-globules were destroyed, and the plasma was stained by the free pigment; it also contained drops of fat and pigment detritus.

Hemorrhages appear beneath the skin, from the nose, mouth, and pharynx, stomach, intestines, kidneys, urinary passages, and genitals.

Gastric hemorrhage is especially dangerous, and is regarded by some as an infallible sign of impending death. The vomited masses often look like soot, and contain, according to Gibbs, epithelium, débris of food, red blood-globules, and sometimes capillaries which are filled with red blood-globules.

Roseola, urticaria, vesicles, pustules, and, in rare cases, herpes facialis may develop.

The temperature and frequency of the pulse again increase.

Some patients are apathetic, others are delirious and die in convulsions; still others have no suspicion of their condition, attempt to leave the bed, and perhaps fall over dead.

The abdomen is often tympanitic, the gastric and vesical region tender.

The majority of patients die in collapse; in some, death is the result of uræmia; in a small percentage of cases, gradual recovery ensues.

Purulent parotitis, multiple cutaneous abscesses, and suppuration of the glands may occur during convalescence.

III. ANATOMICAL CHANGES.—The skin is jaundiced sometimes to a more marked degree than during life. Jaundice is also noticeable in the internal organs and other tissues of the body. Rigor mortis generally appears quickly and is very pronounced.

Hemorrhages are found in the subcutaneous tissue, muscles, epicardium, heart, pleura, lungs, liver, kidneys, stomach, and intestines, urinary passages, meninges, and brain. Transudations or exudations, which may be present, also often contain blood.

The heart muscle is often flabby, brittle, pale, and fatty. The spleen is unchanged.

The size of the liver may be increased, diminished, or normal. It is generally pale and flabby, as in acute yellow atrophy, and the microscope shows marked fatty degeneration. The gall-bladder is either empty, or contains mucoid blackish-green, inspissated bile. The latter is sometimes bloody. Hemorrhages, rarely abscesses, are found in the mucous membrane of the gall-bladder. As a rule, there is no catarrh of the biliary passages, so that the jaundice is probably hæmatogenous, and due to destruction of the red blood-globules by the virus.

The capillaries of the gums and the epithelium of the buccal mucous membrane are in a condition of fatty degeneration. The gastro-intestinal mucous membrane presents hemorrhages, from which superficial losses of substance often take their origin. The intestinal lymph follicles and even the mesenteric glands may be slightly swollen.

The kidneys are swollen, their cortex congested, and sometimes infiltrated with hemorrhages. At a later period, the tubular epithelium undergoes marked fatty degeneration. Hemorrhages may also be found in the mucous membrane of the urinary passages and uterus, and in the ovaries.

IV. DIAGNOSIS.—The diagnosis is easy in tropical regions if the disease is epidemic. It may possibly be mistaken for the following affections: *a*. Bilious intermittent: the spleen is enlarged and prompt effects are produced by quinine. *b*. Bilious typhoid: the spleen and liver are enlarged, and spirilli are found in the blood. *c*. Phosphorus poisoning: recognized by the history, the garlic odor of

the expired air and contents of the stomach, and the chemical demonstration of phosphorus in the latter. *d.* Acute yellow atrophy of the liver: the liver rapidly diminishes in size. *e.* Grave jaundice: these cases not infrequently remain obscure.

V. PROGNOSIS is grave; the mortality in some epidemics was seventy-five per cent. Black vomit, stinking exhalations from the skin, pronounced albuminuria, and anuria are unfavorable signs.

VI. TREATMENT.—Ships, passengers, and merchandise from yellow-fever ports must be strictly quarantined and disinfected.

In the first stage of the disease, we may order mild laxatives, fluid food, and wine. Later, lukewarm baths and stimulants.

Some recommend quinine, others carbolic acid, salicylic acid, and kairin.

PART VI.

INFECTIOUS DISEASES CHIEFLY AFFECTING THE GENITAL ORGANS (VENEREAL DISEASES).

1. *Gonorrhœa. Urethritis Blennorrhoica. Urethral Pyorrhœa. Clap.*

I. ETIOLOGY.—Gonorrhœa is an inflammation of the urethra, caused by a definite form of bacterium, the gonococcus, first demonstrated in the inflammatory products by Neisser, afterwards cultivated and successfully reimplanted in the human urethra by Bockhard, Welander, Chameron, and Bumm.

The mucous membrane of the vagina and neck of the uterus is a very favorable site for the development of the gonococcus. It may develop in the rectum, and cause clap, if the discharge overflows from the vagina, or is introduced unnaturally. The conjunctiva is a favorable spot, and the introduction of the secretion from the urethra greatly endangers the eye. Infection of the mucous coat of the nose and mouth, though reported, is regarded by the most experienced recent authorities as rather theoretical.

Mucous or purulent discharges from the urethra do not always constitute clap, *i. e.*, they are not always due to the gonococcus. Many persons have a discharge from the urethra after being catheterized; others, when a stone has been caught by the urethra and irritated it.

Chemical irritants, as diluted sal ammoniac, may cause inflammation and discharge. So can coitus with women suffering from simple fluor albus, the lochial discharge, the irritating discharge of uterine cancer, or the menstrual discharge. These inflammations are not specific urethritis in our sense of the word. Neither are those discharges of fluid which are described in a few cases of sarcoma of the penis; nor that sticky secretion which comes from the penis in very small amount during violent erection in great sexual excitement. The latter fluid comes from the glands of Littré in the urethral mucous membrane. Purulent discharge may be a sign of a soft, or hard "masked" chancre in the urethra, or urethral polyps, or herpes of the mucous membrane. Gouty persons sometimes have a non-gonorrhœal discharge.

Gonorrhœa is almost always acquired by impure coitus. It rarely occurs in children, usually through violation; in the case of boys, in the rectum. Most of the cases occur in bachelors, especially between 20 and 40 years of age.

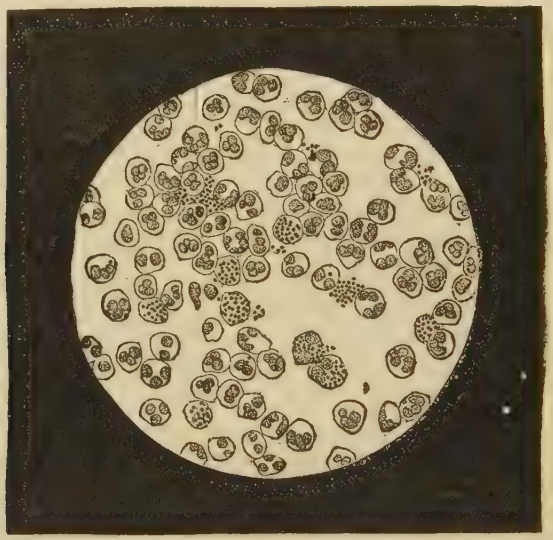
The attempts often made by patients to deceive the physician are ludicrous enough. Some will say it is the sequel of a mere pollution in sleep; others, of taking cold, or bad liquor, or making water against the wind or against the current in bathing, and still more incredible things.

A first attack increases the predisposition to a second. Many have an attack after almost every coitus. One is almost tempted to imagine the existence of a sort of gonorrhœal constitution. Experience shows that when several men copulate in succession with one whore, they do not all get clap. There is least risk when the discharge of semen occurs quickly, and most when it is intentionally retarded, for, the longer the back and forth movements continue, the more abundantly and deeply does the infecting fluid penetrate into the urethra.

The typical locality of the disease in man is the urethra; in woman, the vagina. Gonorrhœa of the female urethra, however, is more common than is supposed.

The affection is at first local, and then spreads to the neighboring parts. Infection of the conjunctiva at birth, when the mother has gonor-

FIG. 42.



Pus from gonorrhœa, with gonococci. Dry preparation colored with methylene-blue. 450 diameters. (Author's observation.)

rhœa, is not uncommon. A man sometimes has several venereal diseases at once, caught either from one person or from several.

II. SYMPTOMS.—Gonorrhœa, like all other infective diseases, has a period of incubation. The first symptoms usually appear from twenty-four to seventy-two hours after coitus, though many authors mention shorter periods: for example, Kühn declares he has seen it begin in six hours; while others still allow a much longer period, as two, three, four, and even eight weeks. Statements going beyond the second week must be looked upon with suspicion. Many patients have the effrontery to fix the date two or three weeks back, when they are not in a position to deny that it may have occurred two or three days before. Others still are drawn by anxiety to consult their physician the very next morning; but such belong to the inexperienced sinners, and learn afterwards to take things more coolly.

Eustache reckoned that the period of incubation exceeded fifteen days only in 35 out of 2,070 patients; he never observed a period of six weeks.

The course of gonorrhœa may be acute or chronic; there is also an intermediate or subacute form. We shall, for practical reasons, describe the disease separately in the two sexes.

Acute gonorrhœa in man, in the majority of cases, is first indicated by a peculiar tickling and prickling sensation in the anterior part of the urethra. This sensation is at first perceived only after urination; it soon becomes permanent, and changes to a painful, burning sensation, while the inclination to pass water becomes frequent, and the act painful.

The lips of the urethra become red and swollen, and if separated, a secretion is seen, mucous in the early stage, and soon becoming purulent. The front part of the urethra, corresponding to the fossa navicularis, is very sensitive to pressure. Drops of greenish-yellow pus soon flow out, either spontaneously or on pressure; these foul the clothes, and leave stiffened spots with marked edges.

If the patient is careful of his person, and diets properly, he may expect the disease, if left to itself, to diminish gradually after the first three or four weeks, and to recover spontaneously from the fourth to the sixth week. The purulent fluid usually grows more mucous towards the close.

It is different when the patient observes a bad diet, or is treated on too irritating a plan. I lately saw a married man suffering in the ninth month most acutely from purulent discharge, in spite of medical treatment. Cases in which the discharge becomes very inconsiderable for a time, or even disappears, and then returns on slight provocation, so that the disease hangs on for much longer than a year, are not specially rare.

Examining the discharge microscopically (with precautions), we find cast-off pavement epithelium and pus-corpuscles, but especially specific gonococci (Fig. 42).

These bodies may be demonstrated thus: Place a little drop of gonorrheal pus between two covering glasses, press the glasses together so that the pus is distributed between them in a very fine layer, and wipe the edges with blotting paper. Then separate the glasses, hold each with forceps, and pass it slowly ten or fifteen times through an alcohol flame till the purulent coat is quite dry. Then lay the glasses with care on the surface of a concentrated watery solution of methylene-blue or gentian violet, in a watch-glass, letting them float if they will, and allow them to remain half a minute. Then take out the glasses with a pincette, and pour distilled water over them; dry them as at first in a spirit flame, and lay them on a glass slide on which a drop of Canada balsam, dissolved in chloroform, is laid. The preparation is now ready for microscopic examination.

The inexperienced observer should take notice that the pus-corpuscles seem enlarged and transparent, so that only their nuclei are distinctly recognizable. The gonococci at once attract attention by their deep color. They are roundish, with sharply marked outlines, large, with diameter about 0.83 μ (1 μ=0.01 mm.). They often lie in pairs, so close together that they might almost be taken for one individual (diplococci). They often lie in masses of ten or twenty, or more, often surrounded by a gelatinous coat, which is best seen in a moderate light, but never take the form of a chain. They often cling to the pus-corpuscles; more rarely to the epithelial cells. They perhaps force themselves into the pus-cells, causing their destruction, by making the nuclei diminish and gradually disappear.

Neisser states that they multiply by the prolongation of the individual, a partition forming in the middle and making two. The same process then takes place in each of the halves, the new partition taking a direction vertical to the first one.

Gonorrhœa has a great many complications, and patients are rarely free from them.

The discharge may become bloody. This is especially the case when venereal excesses occur during the disease; it may also accompany obstinate erections, and excessively frequent pollutions. This form is said to have been common among the Russian soldiers at the time of Napoleon's wars; hence the name of Russian clap. Sometimes the blood is in the form of little streaks, sometimes it is intimately mingled. A change in the coloring-matter of the blood may give a brownish or blackish hue; hence the expression black clap.

Erections are commonly troublesome, and chiefly by night, and in the recumbent posture. They give pain by mechanically stretching the inflamed urethra. Sexual desire is much increased in many, and this favors the occurrence of erections.

Pollutions frequently occur by night, sometimes by day if the fancy is allowed to dwell on sexual thoughts or is excited by bad pictures, reading, and company. The pollutions are painful, because they are attended by erection. They weaken the patient, and keep up the inflammation. I have repeatedly observed a gonorrhœal discharge cease for two or three days and then return after a nocturnal pollution.

Chordee is rather rare, and greatly alarms the patient. It consists in rigidity limited to the posterior part of the penis, the anterior part hanging down flaccid, forming an angle to the other part; the shape is like that of a flail. There is severe pain, as if a cord were stretched through the penis. The cause is probably the pressure of thrombi in some of the cavities of the corpus cavernosum which prevent the distal parts from becoming filled with blood. Circumscribed periurethral inflammations and cicatrices may also form a cause.

A painful desire to pass water is frequent; the urine comes slowly, in a thin stream, sometimes in drops, through the swollen and contracted urethral canal, causing very severe pain.

A great number of complications are due to extension of inflammation to the neighboring parts. First, periurethritis. Here the inflammation attacks the submucous and periurethral cellular tissue, and causes chiefly local points of inflammation, which may be felt as painful swellings along the track of the urethra. The process may go on to the formation of abscesses which may break outwardly or inwardly, or in both directions at once, and gives rise to urinary fistula. In many cases, the abscesses are limited to the follicles of the mucous coat (follicular ulcers).

If gonorrhœal pus accumulates in the sac of the prepuce, inflammation of the glans often occurs, balanitis; or of the inner layer of the prepuce, posthitis; or balano-posthitis in one. These parts are reddened, and a stinking, rancid purulent fluid flows from between them when they are pressed. The smell comes from decomposed sebum preputiale. The patients complain of itching, turning to painful burning when they scratch. Sexual passion is usually increased. A long, narrow prepuce increases the danger of accumulation of pus.

Erosions sometimes occur upon the glans and inner surface of the prepuce; if the surfaces happen to be in contact, adhesions may form between the glans and prepuce which afterward cause pain whenever the sexual act is performed, and cannot be easily remedied by surgical interference.

The prepuce often swells up with inflammatory œdema, becomes red

externally, and cannot be pushed back over the glans. This condition is called phimosis. The orifice may be so narrow that the opening of the urethra can hardly be uncovered. Phimosis occurs much more readily and severely in persons whose prepuce is congenitally narrow. If the swelling and compression of the prepuce go on, they may turn to gangrene unless arrested; black necrotic spots appear, the necrosed mass falls out, and the freed pus escapes through the hole.

Attempts to draw back an inflamed prepuce in phimosis may lead to paraphimosis, popularly known as the Spanish collar; the prepuce springs back behind the edge of the glans and clasps it so tightly that reduction is almost or quite impossible. The prepuce is turned inside out and thrown back. If the glans is not released, it may become gangrenous through compression and anæmia; and the like may occur to the clasping prepuce.

Inflammation of the lymphatics of the dorsum of the penis is not a rare complication. Sometimes one, sometimes both of the chief ducts which accompany the artery are attacked. The back of the penis is painful; a red streak is seen under the skin, marking the course of the lympathic vessel, and a rather hard cord is felt, in places knotty, sensitive to pressure. Abscesses very seldom form. The lymphangitis may sometimes be traced up to the symphysis pubis, and the inguinal glands sympathize, with acute painful swelling. Inguinal lymphadenitis, on one or both sides, also occurs, very rarely with suppuration.

One of the commonest complications of clap is acute inflammation of the epididymis, due to the passage of material which excites inflammation from the prostatic part through the vas deferens to the epididymis. As gonorrhœa generally begins at the front part of the urethra and extends backwards, the epididymis is not usually affected till the second week. Both sides are affected with equal frequency, but seldom both at once. A distinct cause is commonly to be traced: long walks, dancing, gymnastic exercise, riding, or long standing without a suspensory bandage, or tight trousers which mechanically irritate the testes, or too irritating injections, or an accidental blow or squeeze of the testis. If epididymitis has occurred once, it may easily recur in a second attack of gonorrhœa. Varicocele and scrotal hernia predispose to inflammation of the epididymis of the corresponding side. But there are also cases in which a cause is not easily found.

The first symptoms are sometimes general, as a chill or chilliness, headache, dulness of the head, rise of temperature. Some vomit repeatedly. The organ at first feels heavy, and very soon becomes intensely painful, the pain becoming intolerable with every movement, and on standing. The patient stoops and straddles and walks slowly. The epididymis is very sensitive to touch; feels doughy and lumpy, and is enlarged. A free exudation soon occurs in the cavity of the tunica vaginalis propria—acute hydrocele. The inflamed organ swells to the size of a man's fist and more. The half of the scrotal sac is distended, loses its folds, its skin shines and is often reddened, hot, and œdematous—inflammatory œdema. The testes and skin of the scrotum seem grown together. The testis often turns upon its long and transverse axis; if the œdema and hydrocele are great, it may be hard to gain certain evidence by palpation. If the patients take care of themselves, the swelling disappears after about four weeks, the inflammatory fluids are reabsorbed, and retrogressive change occurs. But if injuries are inflicted at the time of the acute inflammation, an abscess may form, though

that is very rare; even gangrene of the testis may be caused by the hydrocele pressing very severely on the testis.

In the favorable case of absorption, a complete return to the former state is rare. Lumpy hardness usually persists, which takes a long time to go away, or may last a lifetime; tuberculosis of the urino-genital apparatus is a quite common result, through bacillary infection and caseous degeneration of the inflammatory deposit. Impotence has been much dreaded in bilateral inflammation, through obliteration of the vasa deferentia. This occurs, but not so often as many think. Atrophy of the testis, mentioned by many authors, can hardly occur except as a consequence of too tight bandaging. Absorption of the fluid may not occur, and the consequence is chronic hydrocele. Neuralgia of the testis after gonorrhœal epididymitis has been seen to occur.

Terillon examined the seminal fluid during acute double epididymitis, and found it purulent, with a large number of granular globules; the spermatozoa diminish progressively in number. He suspects purulent catarrh of the smaller seminal ducts. The spermatozoa may quite disappear, and may remain absent a long time after recovery. In unilateral inflammation, the changes are harder to recognize, as they are partly concealed by the normal semen of the other side.

General symptoms may continue during the height of the developed disease; meteorism, vomiting, even vomiting of fæces, presenting the appearance of ileus; obstinate constipation, giving rise to the suspicion of acute incarcerated hernia. Many patients are tortured by very violent shooting pains in the legs and loins. Others complain of paræsthesia in one or both legs, or slight paresis. The purulent discharge from the urethra generally becomes less or disappears, but usually returns as soon as the acute inflammation is relieved.

Sturgis reports a case in which epididymitis preceded the clap; I have seen a similar one in which I could not detect an error of observation.

Inflammation of the vas deferens, deferenitis gonorrhoica, may accompany epididymitis. The vas is as large as a finger, can be traced as a cord, with various knots, to the inguinal ring, is very painful to pressure, and the skin above it is reddened and often œdematous. Deferenitis without epididymitis is rare. Abscesses may form, and burrow outwardly.

Gonorrhœal prostatitis is a rather rare complication. It often begins with general symptoms. Patients soon complain of a sense of burning and painful pulsation in the region of the perineum. It is difficult or impossible to urinate, and the catheter meets an obstacle. The gonorrhœal discharge usually ceases. Pain is intolerable at defecation, and makes many faint; the stool is intentionally held back, but the suffering is only the greater. The perineum is reddened and swollen, usually warm to the touch, and very sensitive. The finger in the rectum feels the gland swollen, hot, and very sensitive to pressure. Chills sometimes occur, abscesses form in the inflamed gland, bursting into the rectum, or urethra, or though the perineum. There is danger of pyæmia and death.

After an attack of gonorrhœa, the prostate sometimes remains permanently swollen, and prostatorrhœa develops; compare Vol. II., page 350.

Acute inflammation is sometimes propagated to the glands of Cowper

or the seminal vesicles, which may cause abscess. In the former case we find, about half-way between scrotum and anus, laterally from the middle line, a prominent spot covered with red and œdematous skin, hot to the feel, and painful when touched. Pus usually breaks into the urethra. Infiltration of the surrounding connective tissue with pus and urine may cause pyæmia, as in the case of suppurative prostatitis. In inflammation of the seminal vesicles, the finger in the rectum feels swollen and tender bodies at the sides of the prostate. Inflammation not seldom passes from the urethra to the neck of the bladder and the bladder. There is dysury and ischury ; the urine is very rich in mucus, pus-corpuscles, and epithelium from the vesical mucous membrane. Hemorrhage from the bladder may occur. I lately treated a laborer whose gonorrhœa had been cured for several weeks, but who was suffering from very violent hemorrhagic cystitis. The diagnosis is established by finding gonococci in the sediment of the urine.

The inflammation sometimes creeps along the ureters and pelvis to the kidney, causing albuminuria ; the sediment contains epithelium from the mucous membrane of the pelvis and ureters and urinary canals, and casts. Abscesses are sometimes found in the kidney, and uræmia and pyæmia may then cause serious danger.

All the complications here described are mentioned as caused by extension of the disease by continuity. But this does not exclude the occurrence of general disturbances during the course of acute gonorrhœa.

Some patients have a slight fever, especially if found to labor hard. Others soon become strikingly pale, lose strength, can hardly stand. Many are greatly depressed in mind. But a special group of complications is formed by the metastases of gonorrhœa, which, without doubt, are due to the transference of gonococci, contrary to rule, to distant organs, where they set up a fresh inflammation. The only case where gonococci have been demonstrated is that of articular disease.

Shifting, so-called rheumatoid muscular pains are often complained of, and my experience agrees with that of authors who have observed symptoms of acute muscular rheumatism in connection with gonorrhœa.

Many authors describe neuralgia and neuritis, especially sciatica, which last has recently fallen under my own notice. Paretic symptoms are also decribed, which, probably, are connected with neuritis or myelitis.

Fournier lays emphasis on gonorrhœal periostitis, which causes painful swelling of the periosteum, most frequently on the spine of the scapula, os calcis, phalanges and trochanter major, lasts one or two weeks, and sometimes suppurates.

The long-debated question as to whether the occurrence of gonorrhœal arthritis is due to mere coincidence is at last settled by Petrone's and Kammerer's demonstration of gonococci in the inflammatory fluids of the affected joints. Petrone states that he has even seen them in the blood. The attack usually occurs later than the fourth or sixth week of the gonorrhœa, often some time after the purulent discharge has ceased. Acute clap in men is the most frequently combined with rheumatism. The attack may come very gradually, and may be limited to one or a few joints, the knee being one of the favorite points attacked; in other cases it is acute and multiple, and is entirely like acute non-gonorrhœal rheumatism of the joints.

French authors have properly divided the affection into distinct classes. Some cases are gradual and painless, developing without inflam-

mation or larger exudation like hydrarthrosis. In other cases, there are painful inflammatory swellings as in acute rheumatism. In a third class, there is excessive sensitiveness to pressure or movement without local alterations. Cases of acute inflammation with suppuration, pyæmia, and death have been seen; or suppuration, ankylosis, and permanent stiffness of joints. I have repeatedly and recently seen cases of inflamed joints accompanied by purpura or nodes like those of erythema nodosum. Endocarditis, pericarditis, pleurisy, and meningitis occur. Schedler reports a case of death from ulcerative endocarditis after clap. Erythema of the skin and disease of the larynx are reported by Liebermann and Bernier. Phlebitis has been observed. The sheaths of tendons and bursæ mucosæ are sometimes attacked like the joints.

Inflammation of the eye has been observed in patients with gonorrhœa who formerly had the joint disease. The commonest forms are iritis and irido-choroiditis, sometimes associated with opacity of the vitreous, but changes also occur in the cornea. Pauss describes descemetitis; Clemens the same, and also with radiating keratitis.

The following sequelæ of gonorrhœa may be again named: Azoospermia, tuberculosis of the epididymis and urogenital apparatus, chronic hydrocele, stiff and deformed joints, synechiæ of the eye, etc. Pointed condylomata sometimes develop after clap; sometimes at the meatus, usually in the sulcus of the corona, but also at the opening of the preputial sac; they are warty excrescences with many protuberances, sometimes surrounding the entire penis (Fig. 43), caused by the irritating secretion.

Fig. 43.

Pointed condylomata of the inner layer of the prepuce, following gonorrhœa. Author's observation. Zurich clinic.

Chronic gonorrhœa in men is extremely common, almost always due to badly treated or neglected acute gonorrhœa. There are various anatomical changes: chronic granular inflammation, or chronic ulcers of the mucous membrane with granulations that bleed easily, or contraction of the urethra with protracted inflammation behind the stricture. In all cases the membranous portion is chiefly or exclusively affected.

There is not a continuous discharge, but a mixture of purulent fragments, threads, or shreds with the urine, especially in the morning, when the secretion has been accumulating for some time. The meatus is often stuck together in the morning before water is passed, and a drop of sero-mucous rather than purulent fluid will flow out or may easily be pressed out, the pressure beginning as far back as possible and going forward. "Goutte militaire" is a slang term for this affection; another is "hussars' clap."

The microscope shows that these discharges contain pus-corpuscles, angular and shrunken, forming a sort of clot; and pavement-epithelium, often in a state of hyaline degeneration (Fürbringer) (Fig. 44). There are no gonococci.

Many cases of chronic gonorrhœa take the form of fibrinous or croupous urethritis, and fibrinous clots are found in the urine.

If stricture of the urethra is the cause of chronic clap, there will be difficulty in passing water; the stream is weak, twisted, forked, or dribbling; long pressure and much straining are necessary to start it and

keep it going. If there are ulcerations with bleeding granulations on the mucous membrane of the urethra, little streaks or clots of blood may be seen in the discharge.

Chronic urethritis involves many dangers. Patients are liable to become hypochondriacal, collecting every discharge in a special glass to count the fibres, and manipulating the penis to get out the last drop of urine. Many keep up the disease by these measures. The secretion not being contagious, there is so far no reason against marriage. But the danger of a new infection is greater than in the case of well persons, though subsequent attacks are less violent than the first. There is danger from a stricture—or the contraction caused by gradual cicatrization of chronic ulcers in the urethra.

FIG. 44.

Thready discharge in old gonorrhœa. Author's observation. Zurich clinic. 250 diam.

Acute gonorrhœa in women most frequently affects the vagina, vulva, and vaginal part of the uterus, less commonly the urethra; though the latter is oftener affected than is generally supposed. Especially when urine has not been recently passed, pus may often be made to flow out by pressure on the urethra. In acute gonorrhœa of the vulva, the labia are often swollen, reddened, hot, and œdematous, the inner surface thickened, secreting purulent fluid, which is partly dried in thin yellow-brown crusts, and the labia are often stuck together. There is a tickling sensation which excites lust, and often turns to pain. After every discharge of urine, there is pain if the urine touches the inflamed parts.

Swelling, redness, pus, and erosions are also seen in the vaginal and cervical affection. Gonococci in the pus prove the virulent (or rather,

contagious) nature of the discharge. If the urethra is affected, there are frequent desire to pass water, and burning sensations while passing it.

If a woman goes about a good deal while the secretion is free, it runs down and causes eczema intertrigo of the inner surface of the thigh, with redness, moisture, and burning of the skin.

The inflammation sometimes extends to the orifices of the glands of Bartolini, exciting secondary inflammation. There is pain behind one labium, and a tumor is visible, which forms an abscess and may break into the vagina or through the skin. Para- and peri-metritis have been known to occur after acute gonorrhœa; even peritonitis has been described, in which it has been assumed that the morbid agent succeeded in finding a passage through the Fallopian tubes.

Cystitis or nephritis sometimes follows gonorrhœa of the urethra. In other respects, the complications generally resemble those in man. Rectal implication, by the pus flowing down into the anus, is said to take place readily: there is a burning pain in the anus, especially at stool; the folds of the anus are red and swollen, and purulent secretion escapes. The fingers must be free from wounds in examining the rectum; the latter is felt to be hot and swollen, is sensitive, and purulent or muco-purulent secretion is left on the finger.

III. Anatomical Changes.—As gonorrhœa seldom causes death, there are few autopsies. In place of such examinations, much has been observed with the speculum in the vagina, and with the endoscope in the urethra. The latter is a tubular instrument through which light is thrown into the urethra. Desormeux in 1855 made the first thorough studies, and Grünfeld some of the best in Germany. The whole anterior half of the urethra can be seen with an ear-speculum and mirror.

In acute urethral gonorrhœa, the mucous membrane is much swollen and reddened. In some places, vascular trunks full of blood can be recognized; in others, the epithelium is gone; there are small accumulations of pus here and there. The membrane may bleed very easily.

In the chronic affection, the mucous membrane is most commonly swollen, diffusely reddened, and granular—chronic granular urethritis. In other cases, there are ulcers in the membranous part of the urethra, some of which are granulating. Stricture may be added.

IV. Diagnosis.—The microscope renders this easy, for every inflammation containing gonococci in its products is gonorrhœa. Neisser has demonstrated them in the purulent secretion of blennorrhoic conjunctivitis neonatorum, communicated from the mother's vagina at birth.

In diagnosticating chronic clap, we have chiefly to ascertain whether the acute disease has preceded it. As regards the anatomical lesion: the presence of stricture is indicated by certain characteristics in the stream of urine, but the sound is necessary in order to prove its presence. If we cannot prove it, our diagnosis first lies between granular inflammation of the mucous membrane and chronic ulceration; the latter will be inferred if the sound, or pressure from outside, gives sharp pain at any special point of the pars membranacea, or if streaks of blood are seen in the secretion, or if bleeding easily occurs in passing the sound very carefully. The endoscope is not yet in the hands of the profession at large.

V. Prognosis.—This would be favorable in all cases if persons had not the habit of regarding the disease with as little concern as they do a common cold. The result of their carelessness is rarely death, but often

enough the health is impaired for a long time or permanently. The chronic disease often resists radical treatment for a very long time.

VI. TREATMENT.—The best safeguard is to avoid diseased women. The frequency of disease could be much restricted by having women in brothels strictly examined several times a week by physicians, who should be instructed how to make the certain diagnosis by the microscope. The more completely the profession of prostitutes is brought under police control, and private prostitution checked, the less the danger of contagion.

An English physician named Condom is the author of the india-rubber bag used to protect the penis in copulation. Aside from the unnaturalness of this procedure, the danger of contagion is by no means avoided, since the thin coating is easily torn in coitus. It has been recommended to wash the penis in a solution of carbolic acid, two to five per cent, after a suspicious connection; to pass water after coitus, in order to wash out the urethra; and afterwards to inject a solution of two-per-cent carbolic acid, or dilute vinegar, to destroy the virus. Such preventives are not absolutely sure. Another thing to avoid is, visiting houses of prostitution after indulgence in conviviality.

Many plans for cutting short or aborting the disease when it appears have been invented. As a rule, it is said that such treatment must not be applied later than two days from the beginning of the purulent discharge. The remedies advised are nitrate of silver (one part to thirty for injection), or solution of caustic potash, or aqua calcis. This method is theoretically justifiable, but in practice almost always does great harm, for the discharge is not generally suppressed, while severe diseases of the bladder, epididymis, and so on, are readily excited.

Much experience in our own practice has led us to the following plan. The patient lies abed, drinks no strong coffee or tea, eats no highly seasoned food, avoids all sexual excitement through conversation, reading, or pictures, and uses, instead of coffee, milk or weak tea or coffee au lait. Carbonic-acid waters are not to be used. The patient injects a two-per-cent solution of carbolic acid into the urethra every hour in order to get rid of the secretion as soon as possible.

If the disease lasts longer than six days, use an injection of

℞ Zinc. Sulph.....gr. vi.
Aquæ.....fl. ʒ vi.
Iodoformi.....ʒ iiss.
Solve; misce. Shake well before using.

This is to be injected every second hour, directly after using the former injection, which is not to be suspended. But the zinc solution must be retained fully ten minutes by closing the meatus with the fingers. I have cured in a very short time cases which had lasted for months, and had resisted various treatment. The physician must never neglect to warn the patient not to apply his dirty finger to his eye or to other mucous membranes, or sore places.

The syringes that we prefer are the glass ones, with the tip rounded to avoid injuring the urethra. The piston must fit well and draw well. It is filled with the fluid, and held point upward, and the piston pushed forward to expel the air; then the point is slowly and carefully introduced into the urethra, till the nozzle is all within the canal. The thumb and forefinger then press the meatus gently against the nozzle so as to keep the fluid from escaping, and the piston is pressed

very gradually, so that it requires ten or fifteen seconds to discharge the syringe. If the fluid is to remain, the thumb and finger keep the meatus closed while the nozzle is drawn out. Great care in performing these operations is necessary in order to insure success.

Tin or rubber syringes are inferior, because they are opaque and hard to keep clean. Rubber syringes with long nozzles, introduced to the full length, are irritating.

For injection in acute clap a number of astringents or disinfectants have been recommended, none of which seem to be equal to the above treatment. We should in general begin with weaker solutions and increase to stronger ones by degrees. It is advisable to change the remedy once in five days, as the mucous membrane grows accustomed to one. We mention the following solutions: Tannic acid, gr. xv.–lxxv. : ℥ vi.; Crude alum, gr. xv.–lxxv. : ℥ vi.; Acetate of lead, gr. xv.–xxx. : ℥ vi.; Nitrate of silver, or acetate of zinc, or sulpho-carbolate of zinc, or sulphate of copper, gr. iij.–viij. : ℥ vi.; Sulphate of cadmium, gr. $\frac{1}{20}$–$\frac{1}{6}$: ℥ vi.; Subnitrate of bismuth, gr. xv.–xxx. : ℥ vi.; Kaolinum purum pulveratum, gr. lxxv. : ℥ vi.; Corrosive chloride of mercury, gr. $\frac{1}{128}$–$\frac{1}{64}$: ℥ vi.; Permanganate of potash, gr. $\frac{1}{12}$–$\frac{1}{6}$: ℥ vi.; Muriate of quinine, gr. xv.–xxx. : ℥ vi.; Chloral hydrate, gr. xv.–xxx. : ℥ vi.

Insufflation into the urethra has been employed.

If patients will be so foolish as to go about their business while sick, let them at least wear suspensory bandages, which support the testes without compressing them, as pressure might cause inflammation. Standing, running, heavy lifting, and wearing too tight trousers, should be avoided. The internal use of cubebs, copaiva, Peruvian balsam, Tolu balsam, turpentine, may be conjoined with the injection treatment when patients go about.

Cubebs is given with cinnamon or liquorice, a teaspoonful three times a day. Balsam copaiva, best in gelatin capsules, each containing ♏ x., five to ten to be used daily. The others are less active.

Leber recommends tincture of sandal-wood; Vidal, gurjun balsam; and Dupoy, cava (cava-cava or piper methysticum).

When the discharge of pus ceases, the convalescent must avoid excesses in Baccho et Venere for a long time; too early return to beer often produces relapses.

The treatment of complications can only be sketched. For large hemorrhage, raise the penis against the belly and wrap it in cold compresses; if bleeding continues, inject the following:

℞ Liq. ferri perchlorid. . . . . gr. xv.
Aquæ . . . . . fl. ℥ vi.

For erections and pollutions, direct the supper to be taken several hours before bed, and to consist chiefly of fluids. Also give

℞ Potass. bromidi . . . . . gr. xxx.
Lupulini . . . . . gr. viij.
Pulv. camphoræ . . . . . gr. iss.
Morph. hydrochlor . . . . . gr. $\frac{1}{6}$
Pulv. Glycyrrhizæ . . . . . gr. viij.
M. f. pulv. no. ij. S. One before bed-time.

Cold compresses by day for the same trouble. Digitalis is recommended by Bérenger-Feraud.

For chordee, the treatment just described; if induration is felt in the region of the urethra, inunction with iodide of potassium ointment.

For frequent micturition, suppositories of opium or morphine, and not belladona, which often increases the trouble.

℞ Morphini hydrochlor .....gr. viiss.
Ol. Theobromæ.....q. s.
F. supposit. No. iij.

Avoid also the too free use of fluid and carbonic-acid drinks.

Periurethritis requires poultices, and sometimes opening of abscesses.

Balano-posthitis: bathe the penis every three hours in a lukewarm two-per-cent solution of carbolic acid, and insert with an olive-tipped sound, between glans and foreskin, some thread-charpie covered with

℞ Ac. tannici.....gr. xv.
Vaselini..... ℥ ss.
M.

We have also found it very useful to touch the glans and foreskin with lead-water just after washing.

If there is phimosis in addition, inject solution of carbolic acid every three hours with a syringe between prepuce and glans, besides the ointment on charpie. If the phimosis is excessive and inflammatory, use lead-water compresses, and if this does not relieve inflammation, the prepuce must be divided to avoid gangrene.

For paraphimosis, first use cold lead-water compresses, as the trouble often subsides after the inflammation is relieved. If it is thought necessary to relieve it at once, embrace the foreskin between the fore and middle finger, and push the glans back with the thumb of the same hand. If gangrene threatens, incise the inner lamella of the prepuce at the point of tension.

For lymphangitis and lymphadenitis, rub in gray mercurial ointment.

In case of epididymitis, the patient must go to bed. The testis is kept high by a small cushion, and cold lead-water applications are made. All injections into the urethra to be suspended. The bowels to be opened daily with:

℞ Hydrarg. chlor. mit.,
Pulv. Jalapæ,
Sacch. alb .....āā gr. viij.
M. ft. pulv. No. ij. S. Take one powder.

Place three to six leeches around the anus if pain is excessive. If the inflammatory symptoms diminish, wind the testis every day in a wet gauze bandage beginning at the upper part. This is much better than shaving the parts and applying sticking plaster in strips, as it is easy to remove the bandage if it presses. It is also superior to the rubber bandage of Neumann. Scarifications have been successful in case of great accumulations of inflammatory fluid.

For persistent induration of the epididymis, give iodide of potassium (℈ iiss. in ℥ vi. of water, a tablespoonful three times a day), or for anæmic patients iodide of iron, and frictions with ointment of iodine or mercury.

If there are symptoms of vesical catarrh or inflammation of the kidneys, suspend the injections, and give tea of uva ursi leaves, tannic acid, or arbutin; for severe pain in the bladder, warm cataplasms to that region.

In inflammation of the prostate, seminal vesicles, or Cowper's glands, use warm poultices to the perineum; for violent pain suppositories of morphia, or leeches to the perineum, suspend injections, and secure daily stools.

For inflammation of joints, iodide of potash or salicylic acid (gr. viij. hourly till the ears ring), but neither gives prompt relief. Of late, I have often found benefit from pencilling the joints with iodoform-collodium and ice-bladders. Puncture of the joint and drainage may become necessary.

Condylomata acuminata are to be removed with scissors; small ones dry up if pencilled daily with Fowler's solution.

Chronic gonorrhœa is often very obstinate; the list of remedies is enormous. Blackwood reports that the galvanic current applied to the perineum and urethra has proved successful.

The first step is to find the cause. If it is stricture, treat it with bougies, and do not be alarmed if the discharge increases and becomes purulent at first. If it is granular ulceration in the membranous part, use bougies dipped in oil or glycerin, and then sprinkled with tannln, bismuth, alum, or starch. It is better to apply remedies directly to the spot by special instruments; such remedies may be nitrate of silver. In granular urethritis, use same injections as in acute gonorrhœa, only more concentrated. Injections of tannin with red wine and hyposulphite of soda are specially praised.

Urethral suppositories of tannin, lead, lunar caustic, iodoform, or starch mixed with glycerin or cacao butter, are allowed to melt in the passage.

Patients must be warned not to force the discharge out by manipulating the penis; and must be encouraged.

For acute gonorrhœa in women, use sitz-baths, followed by syringing out of the vagina with astringents.

℞ Aluminis..... ʒ iiss.
Iodoformi..... ℈iv.
Vaselini..... ʒ iss.
M. S. To be introduced into the vagina morning and evening, spread on a pledget of cotton.

For inflammation of Bartholini's glands, warm cataplasms and incision when suppuration begins.

2. *Soft Chancre. Ulcus Molle.*

(*Pseudosyphilitic Ulcer. Chancroid* (*Clerc*). *Ulcus Contagiosum Simplex* (*Sigmund*).

I. Etiology.—Soft chancre is a contagious sore, commonly seated on the genitals, and caused by impure coitus. The virus is found in the inflammatory secretion of the ulcer, but its morphology and chemistry are unknown. The ulcer loses its contagious power when it cleans up and begins to heal.

When at its height, the ulcer is very contagious. A single drop of pus has been mixed with half a glass of water without losing its activity. In closed glass tubes, the inoculability remains longer than a fortnight. The dried secretion may be found active if softened in water a long time after. Placed in boiling water, alcohol, concentrated mineral acid, caustic alkali, or astringent fluids, it entirely lost its communicability.

Physicians or midwives may easily get soft chancre on their fingers in examining dirty persons, if there is a lesion of the skin of the finger. Chancres of the nipple, lips, ala nasi, lobe of the ear, eyelid, or hairy scalp are caused by pus reaching those places. In the rectum of man, they indicate unnatural practices, while in woman it may be transferred by pus running down from the genitals. Kissing, the use of water-closets after chancrous persons, the use of drinking cups, eating-vessels, and pipes, foul linen and bandages, and infected wash-tubs are other ways. But such cases are rare compared with those of contact in coitus, and stories are not always to be believed.

It is said that men have lain with diseased women without becoming affected, but have transmitted disease to other women whom they have visited directly after, the explanation being that they have carried infecting pus under the prepuce from one to the other (Ricord, Puche).

The danger of contagion is much greater when there are wounds about the genitals, and wounds may be made during violent coitus in the form of epithelial erosions or cracks of the surface. It is probable that the secretion may penetrate parts where the epidermis is very thin, though there be no lesion.

Soft chancre is more common among men than among women. One often sees a series of several cases at once; and I have repeatedly heard patients trace all the cases to one woman. The soft chancre is seldom found in children; if seated on the genitals or anus, it may be caused by violation; if elsewhere, it may be due to accident.

Soft chancre, like clap, while uncomplicated, is a purely local lesion of the genitals; at farthest, the neighboring lymphatic glands are implicated. The disease is not hereditary, nor does one attack insure against another, in case of new exposure. A coincidence of soft chancre, gonorrhœa, and syphilis is not rare, as one form does not exclude the other. If a man with soft chancre cohabits with a syphilitic person, his sore may be changed into a syphilitic or hard chancre. While his soft chancre, perhaps, heals by degrees, the hard chancre develops very slowly, becomes more and more indurated, and in the place of the former soft sore, a lump of cartilaginous hardness rises, which is afterwards followed by syphilitic changes of the skin and mucous membrane. We must, therefore, be a little careful about prognosis. Such a case is called a mixed chancre (Clerc).

II. Symptoms.—The duration of the incubation is well known from experimental inoculations of well persons. It is so short as hardly to exist. The patients usually say that they noticed the first symptoms on the second or third day, but that proves nothing. Many consider that the period may possibly be from fourteen to thirty days (?).

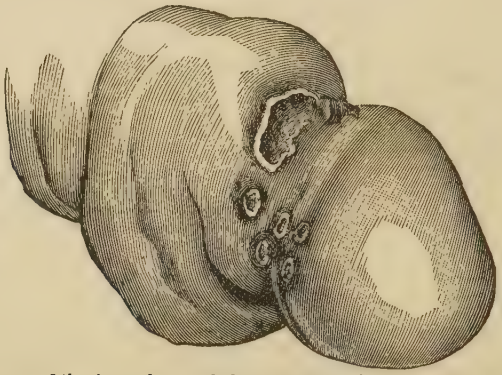
If the inner side of the thigh or upper arm is inoculated with infectious pus, a red border is developed around the puncture within twenty-four hours; in forty-eight hours, there is a red papule, quickly turning to a pustule, with pus under the epidermis; this bursts, and dries to a crust. If this is removed on the

fifth or sixth day, there remains a deep ulcer with the characteristics of a soft chancre. The virus is inoculable on animals.

The characteristics of the soft chancre are usually very marked. It is usually deep, with steep edges, forming a crater ; often perfectly round as if punched out, while at other times its edge has bays, as if eaten into.

The edge is always sharply defined, often a little raised in waves and undermined (Fig. 45). The immediate vicinity is inflamed and red. The surface of ulceration has a yellowish or greenish-gray, tallowy, necrotic, diphtheritic coat, at the period of full development, and the secretion is infectious only as long as this coat continues. The surface is also full of holes, as if worm-eaten. The purulent coat contains pus-cells, and sometimes crystals of ammonio-magnesian phosphate and carbonate of lime. The ulcer cleans itself afterwards, the secretion becomes purulent, the base shows good granulations, and cicatrization occurs; at this period no infectious secretion is produced. If the ulcer is pressed with the finger from one side, pain is felt; prickling, itching, and pain may

FIG. 45.



Multiple soft chancre of the inner layer of the prepuce and the sulcus coronarius. Author's observation. (Zurich clinic.)

be felt spontaneously. The ulcer bleeds easily when compressed or touched with a sponge. The base and vicinity are often slightly hardened, from inflammation, but this hardness passes gradually into the neighboring parts.

In the majority of cases, soft chancres are multiple; the edge of the prepuce, the labia, etc., being fringed with them. Opposite surfaces of skin in close contact often have ulcers of exactly corresponding size and shape; we can sometimes see the auto-inoculation of superjacent skin by an ulcer.

The size varies from that of a pin's head to that of a finger-nail or larger.

The commonest seat of ulcers in man is the external prepuce. They often occur on the edge of the prepuce, its inner surface, and the sulcus coronarius; in the latter case, they may destroy the frænum, or perforate it, or may open the artery of the frænum and cause very troublesome bleeding. Chancrous ulcers may be found at the meatus or in the urethra, but not deeper than the fossa navicularis. In the latter case, a purulent flow occurs from the urethra, and the disease may easily be

mistaken for gonorrhœa. They occur also on the skin of the scrotum, in the genito-crural fold, on the symphysis pubis, and the navel.

In women, soft chancres are most commonly seen on the inner surface of the labia and the posterior commissure of the vagina. The labia are often swollen and red, and the ulcers covered with honey-colored or brown crusts. They also occur on the prepuce of the clitoris, the mons, and the genito-crural fold; more rarely on the mucous membrane of the vagina or the vaginal portion of the uterus. The rectum has been mentioned.

Chancroids usually cicatrize by degrees. The cicatrix is soft, and leaves on the skin spots which are at first pigmented, but afterwards grow white, with occasionally a brown pigmented edge. The ulcer usually begins to clean up in the third or fourth week, and cicatrizes in the fifth and sixth.

The follicular chancroid and the superficial chancroid form a kind of transition to the complications.

The follicular chancroid is a very deep, narrow ulcer, originating in a hair-follicle, and seen with especial frequency in women; the superficial chancroid spreads laterally rather than deeply. The latter sort are common on the glans. Jullien has lately described bullous chancre, which leads to vesicular elevations of the skin. *Ulcus molle elevatum sive luxurians* is a term for cases which have abundant granulations at the time of recovery. Diphtheritic chancre is more serious. Besides a true diphtherial coat over the ulcer, there is often extensive destruction of tissue. The gangrenous chancre is nearly related to this; gangrene, of the ulcer and its vicinity, often spreads very fast, so that in a short time extensive destruction of the penis and skin of the scrotum, the perineum, inguinal region, and abdominal wall occurs. Such cases have been observed with special frequency in hot summer weather, and in anæmic persons, those far gone in consumption, and persons addicted to drink. The use of mercury causes a predisposition to gangrene. Phagedænic chancre also causes rapid destruction without decided gangrene. In these cases, the parts below the skin (as the muscles of the abdomen) may be exposed, as if dissected with a knife. Serpiginous chancre is a term for cases in which a chancre heals and cicatrizes at one point, while extending in another; it is often protracted, and causes extensive ulceration and cicatrization.

Complications may originate from local circumstances, as when the frænum is perforated, or its artery. On the edge of the foreskin, ulcers often have the form of fissures or rhagades, and cause swelling, narrowing of the outlet, and inflammatory phimosis. Ulcers on the inner lamella of the foreskin and the fossa of the glans may also cause phimosis through inflammation and œdema of the prepuce; if the foreskin is drawn back forcibly, this may be converted into paraphimosis. Balanitis also often exists. Ulcers on the glans often go so deep as to destroy a large part of it. Ulcers at the meatus or in the urethra interfere with urination.

Inflammation in neighboring organs often causes complications. Especial attention is due to inflammation of the inguinal glands, which is usually acute, and is designated as acute bubo. This complication is favored by irritating local treatment of the ulcer, or by irritation through bodily exertion, tight trousers, long marches, gymnastics, dancing, riding, etc. Continued excesses with wine and women favor it. The locality has some influence, for buboes often accompany ulcers of the

frænum or fossa, since numerous lymphatics originate in those parts, and their nearest meeting-place is the glands in the groin.

The lymphatics of the dorsum penis are also often inflamed, and then appear as a tender cord, round, somewhat hard, often knotted, the skin over which may be reddened; pus is rarely formed.

Ulcers on one side of the penis cause bubo in the corresponding groin. This is evidently due to the anatomical connections of the lymphatics. Both groins are affected when the ulcers are in the median line, on the frænum, or dorsum of the glans or prepuce.

Buboes may be sympathetic, in which case they resemble those which arise from the presence of any inflammation in the vicinity of the glands. The swelling is usually multiple; the glands tender to pressure, painful in walking, and gradually disappearing when care is taken. Such swellings are always more common in people who are predisposed to glandular enlargment—that is, the scrofulous. They often remain for months or years, and this form has been called strumous bubo.

Chancrous buboes affect only one or few adjacent glands, and have a tendency to acute suppuration. The gland swells, and is often so sensitive that the patient can only walk slowly and haltingly in the stooping position; the skin over it is œdematous, then reddens, and adheres to the gland; periadenitis sometimes develops meanwhile. Suppuration of the gland gives rise to a fluctuating tumor which may burst during any violent movement, or may gradually work its way out through several orifices. In many cases, the pus burrows deeply and may erode blood-vessels in the leg, endangering life by bleeding; or circumscribed or diffuse peritonitis occurs.

The chief point that distinguishes a chancrous bubo from the purulent sympathetic kind is the inoculability of its pus, which can be demonstrated by producing chancroids either on well or on affected persons. It is therefore natural that, after breaking, these abscesses sometimes display such phenomena as gangrene, phagedæna, or a serpiginous tendency. Acute chancroid buboes are sometimes accompanied by very violent general symptoms, as a chill, vomiting, constipation, and fever; I have known several cases in which the physician had thought of incarcerated hernia.

French authors speak of the possibility of the virus passing through the skin without causing a sore, and giving rise to a bubo—*bubon d'emblée*. The point is disputed, and more than doubtful.

Among the sequelæ of soft chancre is destruction of large parts of the sexual organs; in some cases only the stump of the glans or penis remains. Perforation of the frænum is usually of no consequence. Ulcers of the meatus may contract the opening when they cicatrize, which will require an operation for relief. Pointed condylomata may spring up where the external genitals are irrigated with irritating secretions. Fistulous passages may remain for a long time after suppurating buboes.

III. Diagnosis.—Soft chancre is not usually hard to distinguish. We must consider especially *acne*, *herpes*, *hard chancre*, and *cancer*.

Acne is connected with sebaceous follicles, and makes abscesses and ulcers which heal spontaneously in a few days.

Herpes produces a group of vesicles crowded together on a reddened base; the contents are at first serum, which afterwards dries to thin crusts. After the crust falls off, no deep, crater-like ulcer remains. The

trouble heals spontaneously in a few days. Patients often state that they have repeatedly had similar attacks without having had coitus.

Hard chancre, the first distinct symptom of syphilis, differs from soft chancre by its sharply defined cartilaginous hardness; purulent destruction, pain on pressure, and tendency to bleeding are not generally present; the nearest glands show *multiple* enlargement, are not tender to pressure, and have no tendency to suppurate.

Caspary gives the following histological points of difference: In hard chancre, greater deficiency of blood-vessels, greater development of the trabeculæ in which round cells are distributed, numerous open lymph-spaces. Lustgarten's studies assign a greater importance to the demonstration of syphilis bacilli in hard chancre.

A resemblance between soft chancre and epithelial cancer is most likely to occur when parts of the penis have been lost. It will be necessary to review the early history of such cases.

In all doubtful cases, try inoculation (on the thigh), but be sure to make no errors of detail in performing it, and to take the pus at a time when it is yet inoculable.

I have repeatedly seen cases in surgical wards, of extensive destruction of the tissues of the skin, in which it was doubtful whether phagedænic or gangrenous chancre was the cause, or not.

IV. Prognosis.—Though manifold complications may be associated with soft chancre, and may develop into serious diseases, yet the prognosis is usually good. The more care a patient takes of himself in mind and body the better chance of a good sound recovery. Anæmic, poorly nourished, or tuberculous persons are in more danger from the development of complications than robust patients. It is well to keep a patient under one's eye for a time, lest symptoms of syphilis may break out after an unrecognixed mixed chancre.

V. Treatment.—As regards prophylaxis, we refer to the remarks already made in connection with gonorrhœa. Washing the penis and scrotum with five-per-cent solution of carbolic acid after coitus is by no means a sure preventive.

Abortive treatment is of no use unless applied within the first four days after the appearance of the sore. It consists in cauterizing the ulcer and its vicinity so as to destroy all infection. Nitrate of silver, in concentrated solution or in stick, caustic lime, caustic potash, many kinds of paste, and the galvanic cautery, have been used. We are personally averse to this method, chiefly owing to our distrust of the data regarding time given by patients.

Excision of the ulcer is almost always followed by a chancrous state of the wounded surface, which leaves things worse than before.

The treatment of chancroids and the attendant buboes belongs rather to surgery. We mention, however, a few points. Patients must live moderately, avoid beer and wine, have a daily stool, and avoid bodily and mental excesses. Coitus must be avoided. A very thin layer of iodoform is to be powdered over the surface of the ulcer morning and evening, and covered with salicylated cotton spread with carbolic acid, one part, to twenty parts of vaseline. Before each dressing, wash off with lukewarm carbolic-acid solution (2%).

The following dressings are old favorites: solutions of sulphate of copper, ½ per cent; of sulphate of zinc, nitrate of silver, carbolic acid, permanganate of potash, creasote, etc.

In the case of gangrene, phagedæna, or serpiginous ulceration, try to remove the cause (*e. g.*, forbid mercury); give strength by beer, wine, good food, cod-liver oil, preparations of iron or quinine; and employ the above local remedies, or acetic acid with clay (2%). Cauterizing has been attempted. Thiersch recommends for phagedænic ulcers the subcutaneous multiple injections of nitrate of silver (1 part in 1,500); they are inserted at distances of one centimetre from each other and from the edge, but are directed towards the ulcer. Tillot recommended chlorate of potash in the form of a salve (1 : 30).

Phimosis and paraphimosis are treated as before described.

Buboes. The patient goes to bed, applies lead-water on compresses continually, with bits of ice, and lays on the compress a shot-bag or a piece of lead to keep up a gentle pressure. If inflammation continues, use poultices, and at the proper time open the abscess according to Lister's method. We do not think that multiple puncture with pointed bistouries, opening by means of caustic paste, or puncture, is of special advantage.

Developed glandular chancres are to be treated like others. Daily sitz-baths for cleansing the sores are useful. We may try to produce absorption of strumous buboes by painting with iodine or rubbing in ointment of iodide of potash or iodoform, giving also cod-liver oil, iodide of potash or iron, and arsenic.

PART VII.

INFECTIOUS DISEASES AFFECTING THE NERVOUS SYSTEM CHIEFLY.

1. *Epidemic Cerebro-Spinal Meningitis.*

I. Etiology.—Epidemics of cerebro-spinal meningitis can be traced back to the beginning of the nineteenth century. They often attack a small village and spare neighboring places. Epidemics limited to single houses, especially soldiers' barracks, have been known. They usually occur in cold, damp, changeable winter weather, and cease in summer. Children, especially under five years old, are most frequently attacked: after the age of fourteen the disease is rare. The male sex is more liable than the female; and poverty, crowded quarters, and poor food predispose to it. Communication from one person to another is not demonstrated, but many cases are reported in which the disease has been brought into a place by a patient coming from elsewhere, as in the case of recruits (Fraentzel). Many authors, therefore, consider that the disease has only a miasmatic origin, while others term it a miasmatic and contagious disease of infection. An epidemic may last weeks or months, even years; isolated cases may occur for years in a place after a violent epidemic.

At the time of an epidemic, insignificant causes often suffice to develop the disease; I know a case which developed very shortly after the patient had made a dive into the water, without accident. Other infectious diseases are often accompanied by a purulent cerebro-spinal meningitis, *e. g.*, fibrinous pneumonia. (See vol. I., p. 286.) Other epidemics—small-pox, scarlatina, typhoid, relapsing fever, mumps, whooping-cough, malaria, etc.—may prevail at the same time.

A disposition to purulent inflammation of other parts—*e. g.*, boils—is often noticed during epidemics of cerebro-spinal meningitis.

Sporadic cases of the disease may be called spontaneous cases; though the patients often assign such causes as taking cold, over-heating in the sun, mental over-work, or excess of drink, which are unimportant, or at the most only aggravate the complaint.

Nothing is certainly known in regard to the nature of the infectious material. Gaucher, Leyden, and Leichtenstern observed oval micrococci in the inflammatory products, joined in pairs (diplococci), and according to Leyden in chains of from three to six.

II. Anatomical Changes.—The local changes affect principally the pia mater and arachnoid of the brain and cord. There is inflammation (piitis and arachnitis, or collectively leptomeningitis), causing a deposit of exudation, which is commonly fibrino-purulent, more rarely sero-purulent. Death may occur so quickly that at the autopsy nothing beyond severe hyperæmia is found. Secondary alterations of the nervous system also occur; and indications of general infection are very prominent.

The corpse usually decomposes quickly.

On removal of the cranium, unusual tension of the dura mater is noticeable. The bones of the skull are very full of blood. The sinuses of the dura mater are often filled with blood and clots. If the dura is cut and turned back, its inner surface is usually dry, lustreless, and dotted with hemorrhages. In the tissue of the pia and the subarachnoid tissue purulent and fibrino-purulent masses are seen. The veins of the pia, full of blood, are often bordered on both sides by streaks of this material. Purulent secretion is especially common in the sulci of the brain, such as the fossa Sylvii, the optic chiasm, the anterior surface of the pons, and surface of the cerebellum. The tissue of the pia is also swollen, and the sulci of the brain obliterated.

The cortex and adjoining cerebral substance are watery and swollen, and the highly hyperæmic septa of pia mater may often be seen at some depth. Small hemorrhages, lying close together in a group, are often seen. Strümpell described cases of abscess in an epidemic at Leipzig.

The fluid of the ventricles is usually increased, often opaque and flocky, even purulent. Sometimes this condition is limited to a part of the ventricles. Purulent infiltration of the choroid plexus may be associated with the other changes.

The cervical part of the cord is comparatively free; the posterior surface is the part most affected, a phenomenon which has been thought to be associated with the effect of gravitation in a constant dorsal decubitus. The other changes resemble those of the brain—hyperæmia and purulent-fibrinous exudation in the tissue of the pia and arachnoidal trabeculæ, hyperæmia and hemorrhages in the substance of the cord, and rarely an accumulation of pus in the central canal.

The microscope shows that the blood-vessels of the brain and cord are greatly affected. The inner coat and the adventitia are full of numberless round cells, which collect on the outside of the blood-vessel and form purulent streaks, as seen by the naked eye; the same, to a less extent, is seen in the blood-vessels which enter the substance of the brain and cord. The cortex experiences many alterations, especially that of proliferation of nuclei in the neuroglia, and swelling of the ganglion cells and ganulo-fatty degeneration of the finer nerve fibres. In the cells of the ependyma of the ventricles, there is cloudiness, fatty degenera-

tion, desquamation. Inflammatory softening may take place in the adjacent brain substance.

Interstitial inflammation and granulo-fatty degeneration of the nerve fibres may also be observed in the substance of the brain.

De Giovanni has observed hyperæmia of the sympathetic and peripheral nerves, with interstitial nuclear proliferation and fatty degeneration of nerve fibres and ganglion cells.

Cheesy degeneration of the purulent exudation has been described as a commencement of healing. Thickening of the soft membranes and adhesions between them and the brain remain permanently.

The muscles are usually dry, brown-red, pale-yellow in some places. The microscope shows cloudy swelling (Klebs), fatty and waxy degeneration (Rudnew).

The blood is generally of a remarkably dark, berry-juice color, and thick.

The heart usually relaxed; microscopic changes as in voluntary muscles.

Spleen not constantly enlarged, often soft.

Liver and kidneys: cloudy swelling and fatty degeneration have been described.

Stomach: cadaverous softening not rare. Lymph-follicles of the intestine, and mesenteric glands sometimes enlarged and hyperæmic.

III. Symptoms.—The disease often appears in the midst of health without premonition. Prodromata may consist of depression and loss of appetite for one, two, or three days, with nothing specially characteristic.

The outbreak generally consists of a single severe chill; more rarely a repetition of slighter chills. The temperature rises quickly, soon attaining 39° or somewhat higher, though 40° is rare. The pulse increases in frequency, often more than the fever seems to call for, and the same is true of respiration. Violent headache is complained of from the beginning, referred to the front, the vertex, or the back part, or without distinct localization. The pain is overpowering; even when the consciousness is wholly gone, patients sometimes contract the face and press their hands to the head. Giddiness often accompanies the pain, so that the patient totters like a drunken man, grasps objects, and soon cannot stand upright. There is hyperæsthesia of the nerves of sense; patients are greatly annoyed by bright light, and are startled by slight noises.

Drowsiness and coma gradually come on, and with them delirium. During the second period comes the ominous symptom of stiff neck (which gives the German popular name of Genickstarre or Genickkrampf). The back of the head is drawn forcibly backward and downward, and so strongly opposes every effort to bend it forward that we can often lift the constantly stiffening trunk in that way. Forward movement of the head is painful, and even half-comatose patients make a grimace and cry out. In many cases, strong backward bending of the head, and twisting movements, are easy and painless. The head sometimes goes as far back as possible; Hart states that in one case the occiput pressed between the shoulder blades so as to cause gangrene of the skin. The rigidity may vary; I have repeatedly seen it disappear in deep coma and just before death. The explanations of the symptom are various; we think it a direct symptom of irritation of the nerves arising in the cervical region. As the inflammation extends, opisthotonus attacks an increasingly large part of the

spine; patients often lie on the occiput and sacrum. There is great tenderness on pressing the spinous processes—rhachialgia.

In early stages, there is great restlessness, but this soon yields to stupor, in which a person may remain a long time in most uncomfortable positions. Sometimes a short, loud, clear cry is uttered (*cri hydrocéphalique*). The pupils are usually contracted, often unequal, and sometimes dilated in an oval form. The tongue is often dry, cracked, red, even dark with sordes; in other cases, the coating is white, gray, brownish, and not characteristic. The sensibility of the skin is usually excessive, so that slight pinching of the skin causes a loud cry. Vomiting is very common and may be very obstinate. The abdomen is usually sunken, sometimes to the form of a boat or basin, and the abdominal aorta may even be seen beating. The iliac fossæ present deep depressions, marked by the prominence of the crests and spines of the ilium. Traube associates the sinking of the abdomen, not with contraction of its muscles, but with spasmodic contraction of the intestines, due to irritation of the vagus (?). The belly is often sensitive to pressure. Spleen often enlarged, but not uniformly. Stupefied patients often pass no water; the bladder may be full nearly to the navel, and the catheter must be used regularly. Others wet the bed. The urine is scanty and dark, owing to fever, and the small amount of drink taken in the stupor; it almost always contains albumin. Sometimes a large amount is passed, and is light and watery in spite of the fever (urina spastica, vaso-motor and secretory disturbances). Constipation is usual, diarrhœa rare.

The chemical analyses of the urine require confirmation; increase of urea and sulphates and phosphates, and peptonuria, are mentioned (Grocco).

Duration.—Death may occur in a few days; or the disease may last from two to six weeks, with many improvements and relapses, before decided convalescence begins. Great rise of temperature often occurs just before death (to 43° and over); the rise may continue a short time after death. I have repeatedly seen death with symptoms of paralysis of the centre of respiration.

There are special forms—the abortive, the foudroyante, and the intermitting.

Abortive meningitis is indicated by little more than very severe headache, slight dulness, giddiness, and nausea; sometimes there is vomiting and difficulty of moving the neck. There is hardly any fever, and no need to go to bed. All is over in a few days. The symptoms would easily be misunderstood except during an epidemic. Sometimes, however, the disease develops more or less suddenly into the severe form.

The foudroyant attack (m. siderans sive acutissima) comes on with incredible swiftness. Cases are known in which the patient went to work in health, but suddenly broke down and died in a few hours.

The intermittent form is marked by great elevations of temperature at nearly equal intervals, connected with exacerbation of other symptoms.

A quotidian and a tertian type have been distinguished, with exacerbations every twenty-four or forty-eight hours. This is plainly due to the fact that the disease makes progress, step by step, at distinct intervals; there is no reason to infer a relationship to malaria; the spleen exhibits no constant changes, and quinia has no effect.

Complications. The nervous system is often involved. Paralysis may be fully developed, or merely paresis. The facial nerve is often affected,

In the limbs there may be monoplegia, hemiplegia, or paraplegia. Paralysis of the tongue, and dysarthria, or palsy of swallowing, have been seen. In the latter case, we must consider whether the trouble in swallowing is due to the stiff neck. Paralysis may occur very early. Fraser mentions a child that had spasms with left hemiplegia as the first symptoms. General convulsions, or twitching and contracture in certain limbs, sometimes occur. Trismus or attacks of gnashing the teeth are included in this.

The nerves of special sense are often involved. Catarrhal conjunctivitis is not rare; occasionally the secretion becomes purulent. In many cases, important changes in the iris and choroid are added. Chemosis sometimes appears very early, and develops surprisingly fast, usually indicating intra-cerebral pressure and impeded circulation, though it may also indicate that the inflammation has pushed forward from the cranial cavity through the orbital fissure directly into the retro-bulbar cellular tissue (inflammatory œdema). Lagophthalmus sometimes develops, stated by Wilson to be due to rapid shrinking of the orbital fatty tissue and sinking of the eyeball. The oculo-motorius or abducens is often paralyzed, causing strabismus, ptosis, and double vision. Keratitis sometimes occurs, leading to perforation externally and to anterior synechia. Purulent inflammation of the uveal tract is not rare—iritis and iridochoroiditis—causing accumulations of pus in the vitreous and the anterior chamber, and thickening and opacity of the lenticular capsule, and closure of the pupil; separation of the retina may also occur. Neuritis and neuro-retinitis occur, indicating increased intra-cranial pressure. Ziemssen once found apoplectic retinitis. Permanent amaurosis may remain after recovery from the disease. Transitory amaurosis often occurs, connected with temporary disturbances of central innervation.

Rudnew found purulent inflammation of the uveal tract to be the rule in cerebro-spinal meningitis, at least when microscopic examination is made after death. The disease usually begins in the choriocapillaris, and afterwards extends to the entire choroid. Rudnew considers these changes as primary, not dependent on the meningitis.

The organ of hearing is very often affected. There is tinnitus, noises of various sorts in the ear, and deafness gradually comes on; when stupor is present, this cannot be well ascertained. Heller, and Lucæ and Moos found the trunk of the auditory nerve bathed in pus; its neurilemma swollen and hyperæmic; purulent inflammation in the tympanic cavity; hyperæmia, bleeding, and purulent inflammation in the membranous labyrinth. The last may be spontaneous, but the other lesions are mostly propagated along the sheath of the auditory nerve. Many patients are tortured with violent pains in the ear until the tympanic membrane is ruptured and discharges purulent fluid. Meningitis often causes permanent deafness, which involves dumbness in children who had not learned to speak.

The skin is often affected. Herpes facialis often appears on the second or third day; more rarely not till convalescence. It usually begins on the lips, on one or both sides, spreads to the nose, eyelids, and ear, and even covers a great part of the face. It is much rarer on the extremities. Extensive erythema, resembling scarlatina, roseolar and rubeolar exanthemata, urticaria, erysipelas, petechiæ, ecchymoses, ecchymomata, vibices, sudamina, and bullous and pustular eruptions are not rare. They appear at the beginning or the end of the disease, sometimes dur-

ing convalescence. The distribution is sometimes remarkably symmetrical, reminding one of the influence of trophic or vaso-motor nerves. Bed-sores or gangrene of the skin may develop so suddenly that they appear to be due to direct tropho-neurotic disturbances. Multiple joint-swellings have been repeatedly observed, shown by autopsy to be due to purulent discharge or great swelling of the synovial membrane. Kostonopolus, in an epidemic at Nauplia, 1862 to 1864, found joint-affections preceding meningitis.

Catarrh of the throat is much commoner than is usually stated. Diphtheritic changes may occur. Bronchial catarrh is one of the commonest complications; more serious ones are broncho-pneumonia, fibrinous or hypostatic pneumonia, and later abscess or gangrene of the lungs. Pleurisy has been named as a complication.

The respiration often becomes irregular in its rhythm and in its depth as the disease progresses, but pure Cheyne-Stokes respiration is seldom observed. Biot's breathing is more common; respirations of equal depth are interrupted by occasional intervals of apnœa.

The pulse often shows irregularity in the succession and force of the individual pulsations. Irritation of the centre or the trunk of the vagus, from meningeal inflammation, causes retardation of the pulse, while paralysis of the vagus (a much more ominous symptom) causes enormous acceleration. The former is more likely to occur at the beginning, the latter at the close of the disease.

Pericarditis and endocarditis are among the rarer complications; this is true of parotitis, icterus, and glycosuria.

Some of these complications are more properly sequelæ; as paralysis, contracture, deafness, blindness. Permanent mental disorder has occurred. Headache often lingers a long time, and is made worse by mental application and by stooping. Giddiness sometimes continues long after recovery. Leyden described aphasia and anæsthesia. Sometimes there is a peculiar tottering of the body, reminding one of Ménière's disease, and perhaps related to the trouble of the middle ear. I have seen chronic hydrocephalus in a child just one year old, which caused enormous dilatation of the skull. Multiple boils of the skin or muscular abscesses occur.

IV. Diagnosis.—This is usually easy, and rests on the presence of stiff neck, headache, stupor, inequality of the pupils, irregularity of pulse and breathing, vomiting, sunken abdomen, constipation. For tubercular meningitis, see a subsequent section. In children, febrile conditions, especially if connected with intestinal disorder, often cause stiff neck, so that this symptom must be estimated with caution. In deep stupor, the disease might be confounded with typhoid fever or central fibrinous pneumonia. In typhoid, there is meteorism, diarrhœa, an early eruption of roseola, enlargement of the spleen; in fibrinous pneumonia, the sputa are rusty, if any are ejected.

V. Prognosis.—This is always very bad; in some epidemics the mortality has exceeded eighty per cent.

VI. Treatment.—A quiet room, not too light, easily ventilated. Diet liquid; chiefly milk, meat-soup, eggs, diluted wine. If there is stupor, have the bladder emptied regularly three times a day with the catheter. Secure one stool a day, giving, if necessary, two tablespoonfuls of castor-oil in beer foam; or calomel and jalap, gr. viij. each; or compound infusion of senna, nine parts, Epsom salt, one part, tablespoonful three times a day; or clysters. On the head place ice-bladders reaching

from ear to ear; also under the back of the neck. It is very desirable to lay the spine on ice, by means of Chapman's bags. If pain is violent, use repeated injections in the cervical region (hydrochlorate of morphine, 15 grains; glycerin, water, āā ½ oz. M.; ½ of a syringeful).

The following methods are mentioned among many. *a.* Derivatives to the head; rubbing pustulating salve on the shaven head, moxa, painting with iodine, blisters or sinapisms on the back of the neck, cups at the same place and along the spine, the actual cautery, salt or mustard baths for hands and feet, etc. *b.* Antiphlogistics: ice-bladder, leeches to the forehead or mastoid processes, bleeding, inunction with mercurial ointment, ether spray on the back of the neck, calomel internally, nitre, etc. *c.* All kinds of drastics. *d.* Diuretics. *e.* Absorbents, as iodide of potassium. *f.* Narcotics: opium, bromide of potassium, chloral hydrate, ergotin, conium, etc. *g.* Nervines, as preparations of zinc. *h.* Febrifuges: baths, quinine, antipyrin, etc. *i.* Electricity.

2. *Simple Cerebro-spinal Meningitis.*

Meningitis Cerebro-Spinalis Simplex.

I. Etiology and Anatomical Changes.—Purulent inflammation of the pia mater and arachnoid may occur under other conditions than as an independent infectious disease. It is then of a secondary nature, but agrees in its anatomy with the epidemic affection as far as concerns, the membranes of the brain.

The inflammatory changes are often most marked on the convexity of the brain; but the base may be the part chiefly affected. "Meningitis of the convexity" is therefore a false term. "Basilar-meningitis," as a name for the tubercular form, is also erroneous, since the convex portion is almost always affected.

Meningitis may be transferred from another locality. Simple eczema of the scalp and face, and still more erysipelas or boils of the face, are capable of exciting it; it may occur during purulent catarrh of the frontal sinuses. A few cases have occurred after operations on the eye, especially enucleation. It has originated repeatedly from disease of the ear: foreign bodies in the ear, with purulent inflammation, inflammation in the middle ear, polyps, caries and tubercle of the petrous bone. Wounds on the scalp, fracture of the skull, thrombosis of the sinuses, local points of encephalitis on the surface of the brain, superficial abscesses or tumors of the brain sometimes cause it.

Purulent meningitis may originate by metastasis from many infectious diseases if the bearers of infection find their way from the original disease to the meninges. This may occur in fibrinous pneumonia, pleurisy, pericarditis, ulcerative endocarditis, pulmonary phthisis, gangrene or abscess of the lungs, peritonitis, erysipelas, diphtheria, dysentery, cholera morbus, parotitis, typhoid, typhus, relapsing fever, cholera, pyæmia, puerperal fever, septicæmia, rheumatism of the joints or muscles, scarlatina, measles, small-pox, whooping-cough (Bierbaum), etc. It is said to have occurred after vaccination.

Those who believe that suppuration is impossible without the action of lower organisms will at once conclude that every purulent meningitis is an infectious disease in the modern sense. Eberth and Klebs described micrococci in the fluid of the ventricles and the pus of meningitis complicating fibrinous pneumonia. If it is associated with inflammation of

neighboring organs, it may be that the excitants of inflammation find their way through the blood-vessels and lymphatics to the meninges.

II. Symptoms.—The symptoms agree with those of the epidemic form, depending in both cases upon altered circulation of blood, and increased pressure in the cavity of the skull; but in the simple form they are usually slower in their approach, and may be overlooked, owing to the severity of the original diseases.

III. Prognosis and Treatment as in the epidemic form.

B. INFECTIOUS DISEASES WITH VARIABLE LOCALIZATION.

PART I.

TUBERCULOSIS.

Tuberculosis includes all the changes ascribable to the presence of the tubercle bacillus discovered by Koch. Almost any tissue may furnish the soil to develop this fungus, and it is not strange that the domain of tuberculosis is so broad.

The symptoms of tuberculosis in different organs vary so very greatly that they often leave us in doubt. Hence the great diagnostic value of the bacillus, which assures us of the nature of the complaint. Many diseases have been included as tuberculous since Koch's discovery: lupus, fungous inflammation of joints, scrofulosis, etc.

Almost any organ may be the seat of tubercle, but certain ones are far more subject to it than others. The internal organs chiefly affected are the lungs; next the larynx and intestine; then the urino-genital apparatus. In case more than one organ is affected, the original disease was probably situated in one and was transferred to the other.

Solitary tuberculosis, as opposed to tuberculosis of organs, belongs chiefly to the domain of surgery; it includes, *e. g.*, the affection of the joints, bones, single lymphatic glands, etc. It is clinically marked by being often accessible to local treatment, so that by destruction or operative removal of the diseased centre the disease may be cured. It may be that secondary infection will follow in other organs, as tubercle is very variable and mixed in its forms.

General tuberculosis is a third form; also called general miliary tuberculosis; it usually originates from one infected spot or organ, indicates a general affection of the body, and takes an acute or subacute course.

The following pages describe the various forms of non-surgical tuberculosis.

1. *Consumption of the Lungs. Phthisis Pulmonum.*

(*Chronic Ulcerous Tuberculosis of the Lungs.*)

I. Etiology.—A chronic destructive process of the lung tissue, with cheesy degeneration of the morbid products, followed by softening and purulent wasting; excited by the proliferation of tubercle bacilli in the lung tissue. It seems doubtful whether there exists, in addition, a non-bacillary form, as has been asserted.

The disease is very common. Hirsch states that one-seventh of all deaths arise from it; two-thirds of all chronic diseases. It is certainly

favored by modern social modes of life. In factory towns and cities, where the proletariat supports on scanty food its overtasked life, in damp, dark, crowded cellars, it is commonest. But it was known to antiquity, and the writings of Hippocrates contain excellent observations.

Scarcely any disease is so much affected by the influence of constitution. Weakened, feebly resistant, anæmic persons are in special danger. It would seem as if the bacilli were distributed in the air and inhaled by everybody at times, but found no fit ground in sound persons, while in others they easily propagate themselves. Not that they are found everywhere; for they seem to find the conditions favorable to their growth only in human and animal organisms, and at a permanent temperature of 30° (Koch).

A morbid constitution may be inherited, congenital, or acquired.

There can be no serious doubt that pulmonary consumption is inherited in certain families; but it is uncertain what the mode of inheritance is. The fact that it does not always pass directly from parent to child, but often is derived from grandparents or collateral relations, seems to show that a weakness of constitution is transferred rather than germs of disease. In rare cases, the contagious material seems to pass directly from mother to fœtus, causing severe injury to the lungs *in utero*. Demme saw a case of extended phthisical changes with cavities in the lungs of a girl aged 12 days. Berti has found extensive phthisical changes in the lungs of two new-born infants. Johne describes a similar case in a fœtal calf, with tubercle-bacilli in the diseased spots. Landouzy and Martin obtained equally positive results in experiments. In addition to predisposing causes, there must be special occasions for the development of consumption; such occasions, however, occur very often, and only those will escape disease whose constitution resists them with sufficient force. We cannot be too cautious in our views of hereditary consumption; it is so common a disease that it may easily happen that several members of a family are attacked purely by accident.

To hereditary weakness of constitution we must add that which affects children whose parents were debilitated by chronic disease at the time of begetting. Persons having tertiary syphilis or cancer beget children that are weakly from birth, and often fall victims to pulmonary consumption. So with children whose parents married late in life.

Acquired weakness of constitution is very common, sometimes due to improper food and mode of living during childhood, sometimes developing later in consequence of bodily or mental overwork, or of certain diseases. It occurs frequently in diabetes mellitus, owing chiefly to break-down of the constitution, as consumption usually belongs to the late stages of the disease. Onanism, excesses in wine and women, protracted lactations without sufficient rest between assist the development of consumption.

We have stated the frequency of direct exciting causes. Affections of the lungs are naturally the most frequent excitants of consumption; bronchial catarrh or fibrinous or catarrhal inflammation of the lungs may lead directly to consumption in a person predisposed; while in others they run their course quickly and favorably. Symptoms of consumption very often appear some time after recovery from serous pleurisy. Inhalation of dust is often fatal in its effect, hence the bad reputation for producing consumption attached to certain trades. Continuous residence in close rooms may be harmful; consumption is exceedingly common in prisons and pensioners' establishments.

Defects of constitution conjoined with these exciting causes do not form the only way in which consumption is developed. The disease is contagious, and close intimacy and frequent contact with a consumptive person may easily cause the disease in a person not originally predisposed. A long observation of phthisical patients and their families often shows us instances in which a consumptive wife infects her husband, and vice

versa. Years often intervene; the one is long since dead, the other may have married again in perfect health, but the signs of the disease come gradually to view. Consumption was thought contagious in the last century, especially by physicians south of the Pyrenees and Alps (Brisseau); a Neapolitan decree of the year 1783 ordered the destruction by fire of all the clothing of dead phthisical patients.

Contagion is plainly furthered by the sputa, which often contain numberless tubercle-bacilli. Persons seldom care where they spit; the discharge easily dries, and may be transferred to well persons in the form of dust. The material seems not to pass directly through the air from the sick to the well, at least. Celli and Guarnieri could not find the bacilli in the air of hospitals for consumptives.

Villemin, Lippl, Schweninger, and others have shown that animals may be made consumptive by inhalation of pulverized phthisical sputa. A similar experiment has been made accidentally in the human subject. Tscherning reports that a healthy girl injured her finger while cleaning a glass containing sputa abounding in tubercle bacilli; she very soon had a severe tuberculosis of the sheaths of the tendons and lymphatic glands, which made an operation necessary. An observation by Reich deserves notice here; the case of a tuberculous midwife who gave tubercle to ten new-born infants by sucking mucus from their air passages with her mouth after birth. Infection by dried and pulverized fæces loaded with the bacillus is possible, but not demonstrated. Villemin has shown by experiments in inoculation that the sweat is not infectious.

Tuberculosis of the lungs may be due to secondary bacillus infection from other organs. It has long been known that scrofulosis, called tuberculosis of the lymph-glands, leads to consumption, but it has been known to originate from tubercle in other organs. Such conditions cannot always be demonstrated during life, for tubercle, located primarily in the lung, may often develop to so small an extent as to be overshadowed by the development in other organs.

Food may in rare instances be the bearer of infection; for example, milk from consumptive cows or women. Unboiled milk, and raw flesh from consumptive animals, are infectious.

Transportation of tubercle bacilli through wounds of the skin or mucous membrane to the lungs may perhaps occur, but requires more proof.

It has been usually supposed that consumption destroys most victims from the fifteenth to the thirty-fifth year of life, but Würzberg has lately published extensive statistics (relating to Prussia) which show that the highest rate is at from five to ten years, followed by a minimum, after which there is an increase with every decennial period to the end of life.

As regards sex, the frequency varies, being greater for men in Prussia, but somewhat greater for women in England and the United States.

The poor are more affected than the rich; but there is a sort of compensation, since among the well-to-do hereditary influence comes more into play, and acquired disease among the poor.

Climate is hardly of great importance, though there are certain regions where consumption is hardly known, as the high plateau of Mexico, Peru, Costa Rica, the interior of South Africa, Egypt, Iceland, etc. Absolute temperature seems to be less important than dryness of the air and absence of violent changes of temperature. Altitude is unquestionably important. Consumption occurs only by exception in places more than 500 metres high. A change of residence may be fatal; persons in

places free from consumption moving to unhealthy places often are attacked with surprising rapidity Race seems to have less influence, and must be considered with caution.

Dropsy reports that the native peasantry of Galicia are almost perfectly healthy, while the numerous Jewish population die in large numbers about the twentieth year. Early marriages and excessive toil are justly considered as causes.

Much has been said of mutual exclusion between consumption and other diseases—as malaria. This is surely incorrect. Consumption occurs in malarial places; Sangalli reports 144 cases of splenic tumor after intermittent fever, in 25 of which consumption existed (eighteen per cent). The same is true of cancer. Garcin has lately collected sixty-two cases of combination of the two diseases, cancer of the stomach or digestive tube being most frequent. The exclusion between heart disease and consumption must not be overstated; Frommelt especially has shown a frequent coincidence (22 times in 277 cases of valvular lesions). One form of valvular disease (congenital stenosis of the pulmonary artery) almost always has consumption associated with it. (See Vol. I., p. 101.) Alveolar emphysema of the lungs is a rare associate.

Sangalli's thirty-five patients with round gastric ulcer, without a case of consumption, are too few to permit an inference to be drawn.

II. SYMPTOMS.—They usually develop very slowly, often leaving one in doubt for months before decided signs are given. In advanced stages, diagnosis is seldom difficult.

Chlorosis sometimes conceals the first advances. Pallor is conspicuous; the patient is easily tired, complains of heaviness in the legs, of palpitation on bodily exercise, and is often hoarse. Menstruation is often disturbed.

We must be cautious when the patient comes from a consumptive family. Our apprehension is increased when, in addition to chlorosis, there are scrofulous changes—the frequent predecessors and accompaniments of consumption. It is suspicious when a judicious course of iron, in good sanitary circumstances, gives no relief, and pseudo-chlorosis and emaciation persist. Such persons sometimes have a transitory bright redness of the face while taking iron; or the sputa contain small blood-vessels or dots of blood.

In other cases, symptoms of stomach catarrh are prominent at the outset. The patient loses appetite, eructates much, and sometimes vomits frequently; the stool is irregular, and constipation and diarrhœa often alternate. The complexion is very pale and sickly, flesh is lost continuously, there is an increasing feeling of debility, and at last consumptive symptoms are quite manifest.

Consumption sometimes begins with symptoms of frequent and obstinate bronchial catarrh, sometimes generalized and by degrees retreating to the apices, while at other times it is limited to that region from the first.

Catarrh of the larynx is often the first symptom; beginning with hoarseness, continued tickling in the throat, often irresistible impulse to cough, before the diagnosis of consumption can be made. Persistent marked anæmia of the larynx has correctly been remarked as suspicious. Semon speaks of a rapid change from anæmia to hyperæmia.

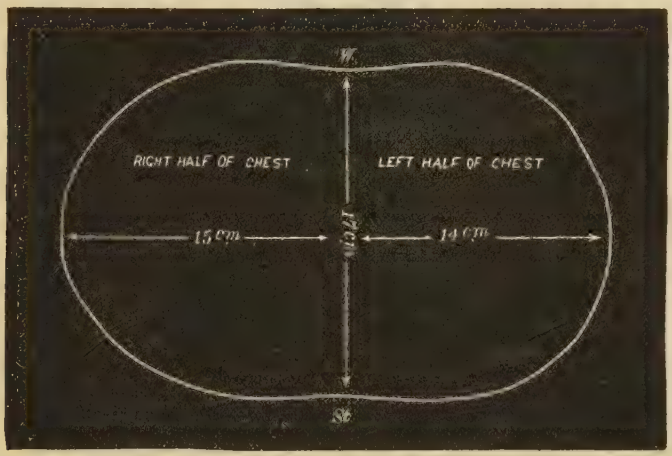
Repeated hæmoptysis, when the first in the train of symptoms, is commonly thought decisive, though years may pass before unquestionable alterations of the parenchyma of the lung are physically demonstrated.

Frequent nosebleed has often been seen in persons who afterwards became consumptive.

Fibrinous or catarrhal pneumonia must be expected to give rise to consumption in debilitated persons; in such case, the upper lobe is known to be mostly affected.

Pleurisy often causes consumption. It is suspicious, when dry pleurisy occurs repeatedly in the region of the upper lobe, because latent phthisis often causes it. Moist pleurisy, without demonstrable cause, running a slow insidious course, sometimes affecting both sides at once or alternately, is usually referable to phthisical changes in the lungs.

Fig. 46.



Cyrtometer curve from a phthisical thorax, at the level of the fifth costal cartilage; one-fourth natural size. (Author's observation.)

We see, therefore, how manifold and insidious the beginnings are, and can understand how great the need of care to avoid errors. We would add that tuberculous disease of bones, joints, and skin, or fistula in ano, or tubercle of the testis are often brought for treatment, and extensive tuberculous disease of the lungs is found on examination.

In advanced stages, consumption is usually easy to recognize, especially when the bacilli are found in the sputa; the local and general symptoms are also generally distinct.

The very appearance of the patient often betrays him; the constitution is seen in the face.

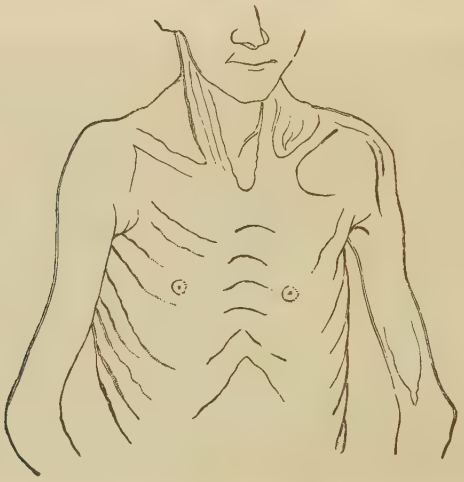
Many patients are persons that have grown tall quickly, and have long necks. The skin is delicate, poor in fat, and strikingly pale; the muscles small; the bones slender. The face is often so lean and sunken that the cheek-bones project sharply. The eyes are sunken, and often have a dark circle around them; they often have a peculiar lustre, and the sclerotica is strikingly blue. The teeth are often long,

bluish-white, transparent, and inclined to caries. The gums have a red border next to the teeth.

The chest is quite peculiar; usually very long and flat, as may be best seen by the cyrtometer curve (Fig. 46). In young persons, it is often very resistant, owing to premature ossification of the cartilages. The intercostal spaces are broad and unusually deep; the angle at which the ribs are attached to the sternum is more acute than normal. The junction of the manubrium and body of the sternum (angulus Ludovici) is unusually prominent, owing to depression of the upper part of the manubrium.

The depressions of the upper part of the chest are extremely deep (Fig. 47). The pectoral and dorsal muscles are usually very small. The shoulders come sharply forward, often giving a marked stoop to the trunk, so that the body threatens to fall forward in rapid walking.

FIG. 47.



Phthisical thorax in a girl aged eighteen years; front view. Author's observation. Zurich clinic.

Seen from behind, the posterior edges of the shoulder blades are raised so that the hand can be partly thrust in under them (wing-shaped, scapulæ alatæ, Fig. 48). This form of thorax has been called paralytic thorax, because supposed to be partly due to weakness of the intercostals, serratus anticus, etc.

The last joints of the fingers are often enlarged (like drum-sticks), and the nails curved like claws. This peculiarity has been explained by deficiency of fatty tissue. The hair of the head is scanty and inclined to fall out.

These constitutional anomalies are not (of course) sufficient to establish a diagnosis, but may be of value in doubtful cases.

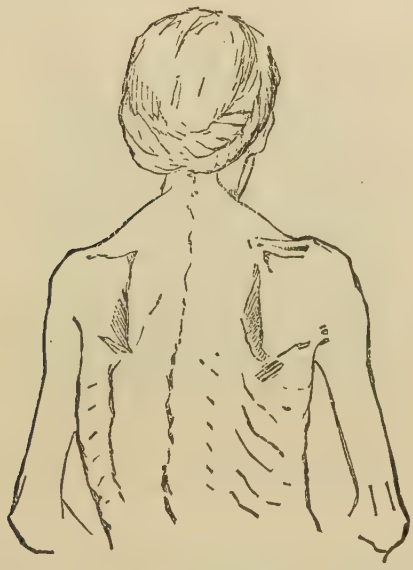
The complexion is usually pale. If the disease in the lungs is extensive, cyanosis occurs, producing a livid tint when slight, but well marked in advanced cases.

The vaso-motor system is very often very excitable. The face, or

the prominence of the cheeks, turns bright red on slight exertion of mind or body, and fever is often marked by the same appearance: the hectic flush at the time of the evening rise of temperature. The flush may be more marked on the side of the diseased lung.

In many cases light-yellow or brown-yellow spots, smooth, shining, non-desquamating, appear on the forehead and upper part of the cheek, sometimes singly, sometimes forming large patches—chloasma phthisicorum—and if Jeannin's statements are correct, hæmoptysis does not occur in these patients, while the spleen and lymphatic glands are often found diseased. In other cases, the skin takes a more diffuse grayish-brown or gray almost light-blackish cŏlor, most marked on the face. Near the end of life the dark pigment often becomes rapidly much more

Fig. 48.



The same, rear view.

distinct. No demonstrable disease of the supra-renal capsules is necessarily associated.

We must not confound with chloasma those light-brown spots characteristic of pityriasis versicolor, which are not shining, and are slightly raised; scales can be taken off by scratching, and exhibit roundish shining microscopic spores, with longish multilocular fungous threads, when examined after adding potash lye, etc. The fungus is the microsporon furfur. (See Vol. III., page 375.)

Pityriasis versicolor is most common on the skin of the breast and lower part of the throat. It begins in spots, spreads over large connected surfaces, and may at last cover great tracts of skin before and behind. Its frequency in consumptives depends on the sweating, which favors the growth of fungi, and on the tendency of the skin to scale, which assists their attachment.

Pityriasis tabescentium is a distinct affection with branny scaling of the epidermis, occurring in many debilitating diseases besides phthisis which produce loss of fat and abnormal secretion of sweat and sebum.

Miliaria or sudamina are not rare—vesicles, clear as water (m. crystallina), or with a little milky turbidity (m. alba), or surrounded by a red areola (m. rubra), usually liberally sprinkled and easily recognized. They show that profuse sweating has occurred, and are seen almost exclusively on the covered parts (breast and belly). M. crystallina is transitory, disappearing when the sweat accumulated between the rete Malpighii and stratum corneum has disappeared by evaporation and absorption. It is often seen in the morning exclusively.

Herpes zoster of the trunk or limbs may occur; I have seen it in a few cases, in which tuberculous caries of the vertebræ occurred some time later. The zoster seemed to indicate a latent inflammation, perhaps pointing to inflammation of the intervertebral ganglia.

Leudet found seventeen cases of zoster in one thousand of consumption. He often noticed a combination of disturbance of sensibility and motility, and thinks protracted cases are the ones chiefly affected.

Sweating is very common. A patient's skin often becomes moist, or covered with drops of sweat when he is excited or tired. The sweating is often confined to the night, especially between midnight and early morning. It is very profuse, weakens the patient, and is well known as hectic sweating. There sometimes is a very penetrating odor of fatty acids.

The cause of sweating is not certain. Some connect it with fever; but fever is not necessarily present when the sweating exists. Others assume general relaxation of tissue as an explanation. Traube suspected that the skin excreted water vicariously, as the lung performed that function imperfectly. Lauder Brunton says that the respiratory centre being depressed in activity, carbonic acid may accumulate in the blood to such an extent as to irritate the centre for sweating.

I have seen a few cases of unilateral sweats; the patients had symptoms of caverns on one side, corresponding to the side of the sweating (implication of the sympathetic?). See Vol. III., p. 348.

The fat almost always disappears from the skin, which is raised in folds, transparent and thin, like paper. But I have in a few cases noticed a good, even an unusually good development of the panniculus adiposus in spite of extensive disease of the lungs and general loss of strength.

If the patient has remained long in bed, bed-sores may appear, especially if frequent change of position and a smooth surface to lie on have been neglected. The commonest position is the sacrum, owing to the pressure and the superficial position of the bone; then the heels, trochanters, malleoli, and shoulder blades. Such occurrences are very unfavorable, paining and weakening the patient and giving trouble to the nurse.

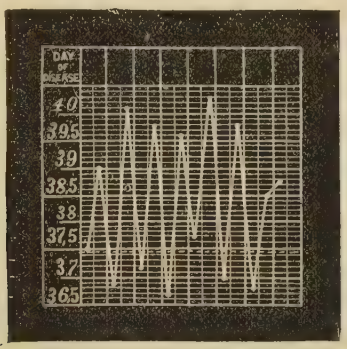
Many patients attend to their business quite regularly in spite of advanced disease. Others have occasional bad turns, which keep them in bed, caused by changes of weather or taking cold. Others remain in bed for months or years. In advanced cases, especially if want of breath is felt, patients prefer to have the body raised in bed; the choice between

side and back is a matter of individual preference, though inflammation of the pleura of one side may lead the patient to lie on the other side.

Extraordinary development and activity of the mind is often observed. Wise children are popularly said to die young. In the late stages of the disease, mental power is usually retained: it is characteristic to see the mind cheerful and courageous, often busy with plans, while the body is slowly perishing. Delirium is very rare; it is almost always a very unfavorable sign, indicating the approach of death.

Fever is almost always an accompaniment, though there may be long periods free from it. The rise of temperature is often very slight; but very high temperatures may occur at noon or evening, often preceded by a slight chill with great pallor of skin. If the morning temperature is normal or beneath normal, while the evening heat is excessive, we have hectic fever (Fig. 49), due, as it often is, to absorption of purulent masses. Continuous high fever is especially common in very rapid cases (phthisis florida).

Fig. 49.



Temperature curve in the final period of consumption. Hectic fever. (Author's observation.)

Inverted types of fever with the greatest heat in the morning are not rare, as Traube showed. Brünniche says he finds it chiefly when there is associated miliary tuberculosis.

Peter and Vidal state that the skin temperature is raised over caverns and infiltrated spots, and that the thermometer can be used to define the diseased district. I have not found it so. McAldowic found in cases of unilateral disease that the temperature in the axilla of the sound side was higher than in the other as often as the contrary; but if infiltration existed on one side, and caverns on the other, the former side usually had the higher temperature in the axilla.

The pulse gives no characteristic signs; it is usually accelerated: as emaciation and weakness increase, it becomes small and soft, and in febrile states dicrotism is usually quite striking.

Dyspnœa may give no trouble during the whole disease, probably because the wasting body requires less air and gets used to less. In bronchial catarrh, or if the disease becomes acute, it is generally felt; febrile states may excite it.

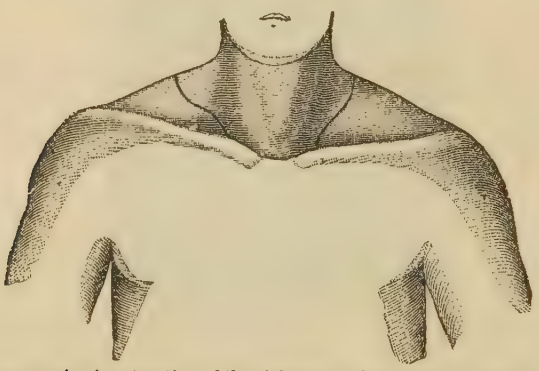
Œdema is one of the late symptoms. It may be simply due to marasmus, or may form a symptom of nephritis. It usually begins in the lower extremities. If patients are obliged to lie chiefly on one side, œdema of the subcutaneous cellular tissue affects one side chiefly or solely. In one extremity it may be caused by marantic thrombosis.

Local changes in the lungs almost always begin at the apex, and often are wholly confined to the upper segment of the lung.

The cause of this may be that the apices are the parts least used in breathing, whence come opportunities for deposit and long retention of inflammatory products, especially in the alveoli. In addition, this region has a poor supply of blood. In persons of consumptive habit, the poor development of the upper chest-muscles, and the early ossification and deformity (Freund) of the upper costal cartilages, still further impair the movements of the chest and the expansion of the lungs. The arms, attached to the thorax, have also a normal power of restricting the excursions of its upper part.

The local changes in the lungs may long consist solely of catarrh of the apex. Rough vesicular breathing, interrupted vesicular breathing, decided prolongation of expiration localized at the apex, or unequal intensity of respiratory murmur at the two sides are often the first and only signs. These symptoms are especially important when confined to one side and varying in intensity: if on both sides, they may not denote phthisis. Sibilant rhonchus or single moist crepitant râles may occur.

Fig. 50.



Boundaries of percussion in retraction of the right apex; front view. Author's observation.

In forced breathing, single, clear, and clicking crepitant sounds may occur, due to contraction of the chest-muscles.

Eccentricities in the respiratory movements are often connected with the above symptoms. Parts of the thorax where respiration is abnormal may be seen—or more readily felt with the flat of the hand—to move less actively than other parts.

Haenisch has made a special instrument to measure the excursions of the regions of the chest.

Abnormal percussion sounds are often the first signs; slight dulness in the upper fossa on light percussion, and increased resistance to palpatory percussion.

Slight differences often occur in health; a fuller development of the muscles on the right side often gives a greater dulness on percussion.

A very important sign is the difference in the height of the apices,

shown by percussion, as was first stated by E. Seitz. This symptom indicates simply contraction of the apices, but it is a specially frequent accompaniment of chronic tuberculosis of the lungs (Figs. 50, 51).

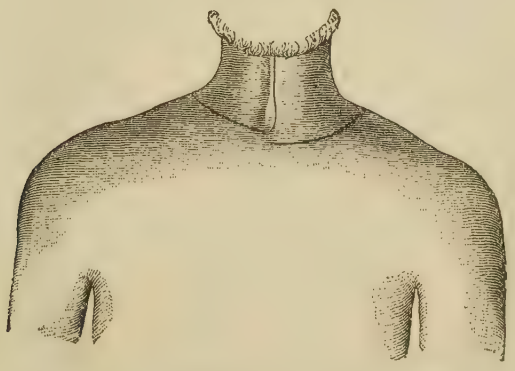
When consumption is allied with extensive contraction of the lungs, the thorax is retracted, sometimes in the upper fossæ, sometimes over one whole side.

The diagnosis is usually certain when there are signs of extensive infiltration or caverns in the lungs; the symptoms agree in all points with those of similar conditions due to other causes.

Infiltration of the alveoli with firm, usually cheesy masses is indicated by increased vocal fremitus, dulness, bronchial respiration, increased bronchophony, and consonance of crepitant râles which may be present. Williams' tracheal tone may also be formed.

Cavern-symptoms are distinct in proportion as the cavern is near the surface, and is large and smooth-walled. If situated deeply, they require stronger percussion. Over those of the size of a walnut we hear a

FIG. 51.



The same, from behind.

tympanitic percussion-sound, bronchial breathing, and tinkling crepitus. If the cavity is about six centimetres in size, with smooth walls, metallic symptoms occur, both on percussion and auscultation (cracked-pot sound, increased vocal fremitus, bronchophony), but only when the cavity contains air. If quite full of secretion, there is dulness; and the alterations from dulness to tympanitic sounds may be a very important fact in diagnosis.

The sound of a falling drop, and that of succussion, are rare. Consult special works for Wintrich's variation in pitch; the interrupted form of the same; Gerhardt's variation in pitch, and the respiratory variation in pitch.

It is not always easy, though desirable, to make a certain diagnosis of caverns. E. Seitz gave as a sign the metamorphosing respiratory murmur, though Botuwschikoff states that he has observed it in fibrinous pneumonia. Baas gives post-expiratory crepitus as a sure sign.

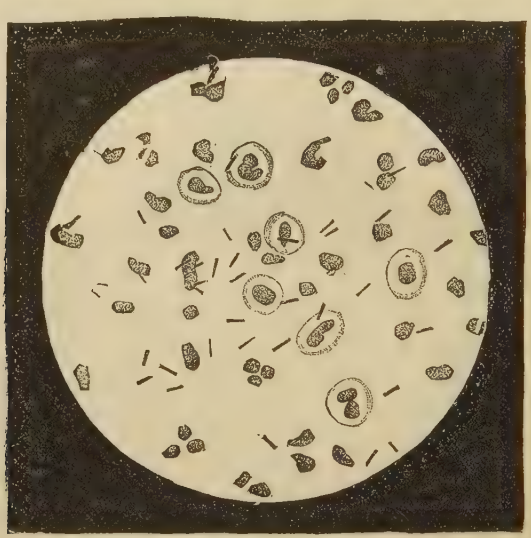
Metallic symptoms have repeatedly been heard over smaller cavities (Kolisko, Wintrich), but it seems necessary that the cavern should be superficial, regularly formed, and especially smooth-walled, should lie near a large bronchus, and be connected with a bronchus with wide opening. Symptoms which are connected with the movement of the heart, especially systolic crepitus, may exist over caverns. Peculiar roaring or hissing sounds are sometimes heard (von Brunn,

Schrötter), apparently originating in the blood-vessels, which project partially into the cavity, and have their resonance increased.

The sputa are of great and special importance. The demonstration of tubercle bacilli is decisive, for they occur in the sputa of no other disease. In a few cases of undoubted consumption, they are not found in the sputa after repeating the attempt several times a day; this happens very rarely. I have repeatedly succeeded by letting the sputa stand twenty-four hours, and trying again.

The bacillus may be demonstrated in the following manner: Take from sputa which certainly come from the lungs and not the upper air passages, a piece of the size of a pin-head, and distribute it on a perfectly clean covering-

FIG. 52.



Tubercle bacilli from the sputa of a consumptive patient. Fuchsine preparation. Immersion, 750 diameters. (Author's observation.)

glass with a carefully cleaned platinum wire or needle. Lay on it another perfectly clean cover-glass, and press the two carefully together so as to distribute the sputa uniformly in a thin layer. Wipe the edges, if necessary, with blotting-paper. Draw the glasses apart sideways, let the material dry half a minute; then take each glass in a pincette, and pass it through the flame of an alcohol lamp or Bunsen burner from four to ten times, about as fast as one cuts bread, holding the sputa upward.

The coloring fluid is best prepared fresh every time. A concentrated alcoholic fuchsine solution, pure colorless aniline oil, pure nitric acid, and solution of malachite-green are kept on hand. The oil, originally almost as clear as water, turns when exposed to light, and had best be kept in a box.

Pour into a clean test-tube enough aniline oil to fill the bottom, then add water till four-fifths full. Close the mouth with the thumb, and shake vigorously until a partial emulsion of the oil is produced. Filter, and collect the clear filtrate in a large watch-glass. Add to the clear contents of the watch-glass five to ten drops of alcoholic solution of fuchsine, and place the two covering-glasses, sputum downward, so that they float on the fluid. Cover the watch-glass with another, to keep out dust, and let it stand twenty-four hours. If necessary to

finish quickly, heat the solution (as Rindfleisch proposed) over a flame until bubbles begin to form; let the cover-glass lie in it ten minutes, and prepare it for microscopical examination. We prefer the former way.

The next step consists in filling a watch-glass with pure absolute alcohol, adding to it by means of a glass rod a drop of pure officinal nitric acid, and mixing carefully. The two cover-glasses are laid in this with the sputum side up, when the diffused fuchsine tint will quickly depart, and by degrees the glasses grow pale. When the color is gone (or, if there are many bacilli, nearly gone), rinse them quickly in distilled water. The bacilli now show the staining, while the other parts are colorless. A pretty contrast, useful for a beginner, is made by coloring the other parts with another tint; we prefer malachite-green as giving a fine contrast. For this purpose, lay the glass one minute in the malachite-

FIG. 53.

Tubercle bacilli with spores from the sputa of a consumptive patient. Author's observation. (Zurich clinic.) 750 diameters, immersion.

green solution, face downward, raise it with the pincette, and wash off again quickly in distilled water. Then press both surfaces gently on clean blotting-paper to remove the water, draw it several times through a spirit or gas flame, with sputum side up, and finally treat with chloroform-Canada-balsam. Place a drop of the balsam on a clean slide, and lay the cover-glass, sputum down, on the drop; the later spreads equally under the glass. In most cases, ordinary lenses of three hundred to five hundred diameters suffice, but in doubtful cases we require Abbe's apparatus for illumination and oil-immersion lenses.

The tubercle-bacillus is a rod, sometimes straight, sometimes bent at an angle, from $1\frac{1}{2}$ to $3\frac{1}{2}$ μ long (1 μ = 0.001 millimetre), equal to about one-third or one-half of the diameter of a red blood-corpuscle. They

are often very numerous, and distributed with much uniformity in the preparation (Fig. 52), while at other times they are grouped, and so crowded that the groups can hardly be analyzed into their elements (Fig. 53), or they are single, and careful inspection is required to bring to view one example. Sometimes one glass shows some, while the other does not. On successive days, the number may vary greatly. They are usually numerous in proportion to the acuteness of the process in the lungs. Fine globules are often found inside of the bacilli which do not take the aniline stain; they are spores (Fig. 53).

The chemical examination of sputa is not of great importance. Renk estimated the daily sputa of three patients at the average of one hundred and twenty-four grams. The average composition by percentage was as follows:

	Case 1.	Case 2.	Case 3.
Water	94.58	94.97	93.84
Solids	5.42	5.03	6.16
Organic components	4.66	4.12	5.36
Inorganic components	0.76	0.90	0.80
Mucin	1.80	2.56	2.84
Albumin	0.49	0.11	0.29
Fat	0.36	0.30	0.52
Extractive matter	2.01	1.16	1.71

The phosphates preponderate among the inorganic components; they may amount to two grams daily (Stokvis).

One who constantly examines the sputa will very often find elastic fibres, constituting evidence of consumptive disease, at a period before the other methods of physical examination can give much information. At later periods also, when the disease is well marked, we should never omit to examine for elastic fibres, as they indicate the activity of the destruction of the lungs and the effects of treatment.

Elastic fibres are easily recognized by their curled, sharply outlined, often divided form (Fig. 54). The addition of potash solution causes the cell-components to disappear, while the elastic fibres become more distinct. They are distinguished from crystals of fatty acid by not dissolving in ether or boiling alcohol, not melting when warmed, and showing no varicose swellings when pressed. The fatty acid crystals are dissolved by long treatment with caustic alkalies.

In consumption, the loss of tissue is almost always very gradual, and the particles in the sputa are generally microscopically small. Visible fragments (as seen in abscess or gangrene of the lung) are very rare. Hence it requires both patience and skill to find elastic fibres.

In microscopic work, select very small bits, and work them out finely; pay special attention to peculiarly opaque and slightly grayish spots, which often contain elastic fibres.

Fenwick gives a convenient and certain way of finding elastic fibres, which we repeat with a slight practical change. Put the sputum into a beaker glass with an equal quantity of distilled water and as much of a solution of caustic potash (1 : 3). The thickish gelatinous mass is heated to boiling, being constantly stirred meanwhile, which makes it perfectly fluid. The glass is allowed to cool, the clear fluid is poured off from the sediment, the latter is poured into a pointed glass, and the sediment having settled again, is taken by a pipette for examination. The method is very accurate, and not only discovers the fibres, but enables us to estimate their number.

Sawyer recommended caustic soda instead of potash, other points remaining the same.

Remak's statement that elastic fibres from the bronchial mucous membrane are distinguishable from those of the lung-parenchyma by being finer, is not of practical use.

The presence of alveolar epithelium in the sputa is of much less importance. It has diagnostic value only when very abundant, and when there are also signs of apex-catarrh. The cells are usually roundish or roundish-angular, and in the condition of fatty or myelin degeneration; partly broken down, so that little granules of fat and drops of myelin have been set free (Fig. 55). They often compose almost the whole of the sputa. They are blackened by osmic acid, like all fatty substances. They may be taken as showing that active throwing off of epithelium is taking place in the alveoli, with degeneration of the cells.

Cholesterin-crystals, sarcina, and mould-fungi are of less consequence. For the latter, see Vol. I., p. 326. Phthisical changes due to inhalation of dust may exhibit the dust free or inclosed in cells. We give a view from Traube, showing pneumoconiosis anthracotica (Fig. 56).

Fig. 54.

Elastic fibres from the sputa of a consumptive patient. (Author's observation.) 275 diameters.

"Lung-stones," expectorated calcareous concretions, are not of great importance. They may be calcified parts of lung, when the addition of muriatic acid will restore the outline of the tissues (Rindfleisch). Kloman also found in them elastic fibres, lung-pigment, granular detritus, and tablets of cholesterin. Many consumptives cough up such lumps so frequently as to give rise to the term "phthisis calculosa."

Lime concretions sometimes originate, not in lung-tissue, but in cretaceous changes of bronchial glands which afterwards break through into the air-passages. They may be so large that they cannot pass the vocal cords, and suffocate the patient (Rühle).

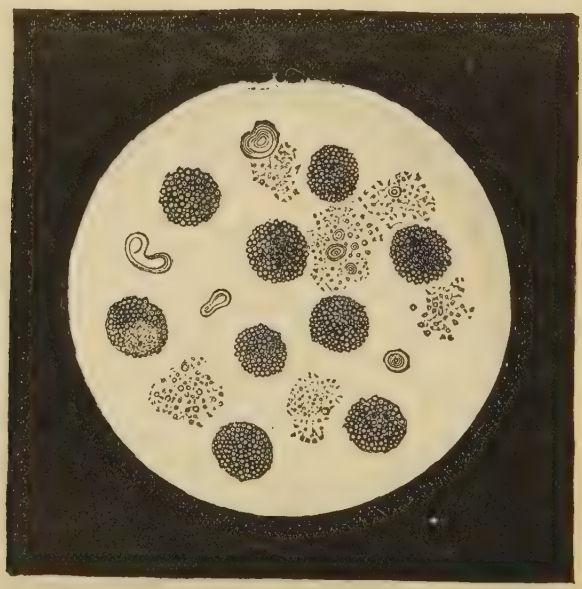
Bone-formations occur in the sputa; originating in the lung, or passing from a vertebra into the lung (Charon).

The gross appearances vary greatly. At the beginning of the disease, we often have a tough, glassy, slimy, transparent sputum, which differs little from that of bronchial catarrh. It is sometimes gelatinous, or

like frog's spawn, which indicates a similar alteration in the lung. If consumption follows fibrinous pneumonia, the expectoration is often green (Traube and Nothnagel).

The more extensive the disease in the lungs the more abundantly is pus found in the sputa, which may often be chiefly purulent. If cavities form, the sputa often take a distinct characteristic form, either that of coin (sputum rotundum, nummulare, sive nummulosum), or that of balls (s. globosum). Nummular sputa consist chiefly of opaque green purulent matters, found at the bottom of the spit-cup in the form of regular round lumps, usually sharp-edged. The globular sputum is a roundish, gray-green, separate ball of pus, with a torn and ragged surface, sometimes kept floating by air-bubbles, sometimes sinking to the bottom of the glass.

FIG. 55.



Fatty alveolar epithelium cells; some full of lung-pigment, some broken down. Also, myelin forms. 275 diameters.

The peculiar form of these sputa is due to the cohesion between the particles of pus. If sputa are collected in water, a crumbly sediment is often deposited; if the sputa are very thin, the same occurs without water. Exactly similar masses are found after death on the walls of cavities, and Virchow long ago showed that they proceed from the purulent decay of lung-tissue. They form no unimportant sign of cavities; they often contain large numbers of the bacilli.

Bloody sputa form a very important sign, often giving the first indication of consumption; though not every one that has had hæmoptysis becomes consumptive. The demonstration of the bacilli in the bloody spit is decisive. The sputa may be pure blood, or tinged with blood. In the later course of the disease, severe hæmoptysis may occur repeatedly. It is either provoked by bodily and mental exertion and excite-

ment, or occurs spontaneously. Very profuse bleeding may proceed from the rupture of aneurisms of arteries in the walls of caverns. Rasmussen has studied this form of bleeding closely, and Fraentzel has added observations.

Gerhard and Brehmer have lately noted intermittent hæmoptysis, occurring at certain hours of the day, and relieved by quinine. In Gerhard's cases, it occurred at night at the apyrexia, while Brehmer saw it also at the height of the fever.

The frequency of hæmoptysis is usually exaggerated. Among 369

Fig. 56.

Sputa in pneumoconiosis anthracotica; after Traube. Magnified 290 diameters. *a*, Tüpfelzellen.

cases, Condie found only 87 (24 per cent) at any time of the disease; in 11 per cent it formed the first symptom. Williams' figures are considerably larger; 70 per cent, or 569 in 1,000 cases.

Expectoration is difficult, especially at the beginning of the disease; the irritation and the effort involved in coughing are a torture, the sleep is disturbed, and there are pains due to the violent contraction of the muscles of the chest and abdomen. At a later stage, expectoration is apt to be easier, and usually more copious.

In a few cases, there is very little cough and expectoration, and the physical signs over the lungs are very slight, in spite of extensive alterations of the

lung-parenchyma; but the wasting of the body should arouse suspicion and prevent error.

The heart is very often involved. The second (diastolic) pulmonary sound is often intensified, a sign of increased blood-pressure in the distribution of the pulmonary artery. If there are signs of retraction of the lungs, the heart may be dislocated. If smooth-walled cavities lie near the heart, the sounds of the heart sometimes receive a metallic character, through resonance.

The subclavian murmur, described by Stokes, is thought very important by some authors. Rühle considers the subclavial expiratory murmur a valuable diagnostic sign.

It is heard during expiration as a hissing or roaring sound with the systole. It has been explained by pleuritic adhesions excited by lung-changes, which have involved the subclavian artery, and thus cause bending and narrowing of the arterial tube during the movements of respiration. It is found in healthy persons.

There are no characteristic peculiarities in the urine; its amount is commonly lessened, and the urea and chlorides are usually diminished. Some authors affirm an increase in the phosphates (Teissier), but Stokvis rightly denies this, and found them usually diminished. Stokvis, opposed to Beneke, found no characteristic change in the earthy phosphates. Senator mentions increased excretion of lime. Traces of albumin may be found in very exhausted patients. Vibert found sugar three times in fifty cases.

All the other functions may be unaltered, though such is not the rule; consumption very often has complications, and almost every organ can be involved.

The bones are often carious—tuberculous caries. The extremities, spine, skull, may be the seat of this caries. In the skull, the petrous bone is chiefly attacked, causing loss of hearing and paralysis of the facial nerve; or thrombosis of the sinuses, meningitis, or abscess of the brain. The presence of tubercle bacilli in the discharges from such disease demonstrates its nature.

Tuberculous (fungous) joint inflammation is closely related to tuberculous processes in the bones, though more properly pertaining to the domain of surgery.

In very weak patients, the muscular excitability is often increased. If we give the pectoral muscle a light blow with a percussion-hammer, the spot struck rises, and remains so for some seconds. Graves and Stokes described the phenomenon, and correctly stated that it is not pathognomonic of consumption, but occurs also in other states of weakness Tait has studied it carefully, and calls it myoidema. Without assenting to all that he says, I have often seen (as he states) that it sometimes occurs on one side only, and is apt to be stronger on the side most diseased.

The peristaltic contraction (best described by Auerbach) is essentially different. In this case, when a muscle is tapped, there is a rising on both sides of the spot struck, which gradually extends in a slow wave to the two attachments of the muscle. This form is not frequent; is sometimes limited to single muscles; is not characteristic of consumption, but exists in the marasmus of gastric and intestinal disease, and probably in many other weakened states. The symptom is often unilateral in these cases also. In a case lately seen by me in König's clinic, the muscles were found microscopically unaltered.

Tuberculous inflammation sometimes occurs in peripheral lymphatic glands; they swell and are hard, but may afterwards soften and become purulent, burst, and lead to protracted sinuous fistulæ.

The larynx is very often attacked. The trouble may be purely functional—obstinate hoarseness, either without anatomical change, or caused by paralysis of the vocal cords. The latter is probably due to the atonic state of the muscles of the larynx, dependent on general marasmus. Permanent hoarseness is, however, more commonly due to catarrh, which may be limited to one side, or to one of the true vocal cords—not always on the side of the lung disease.

Tuberculous ulceration of the larynx (laryngeal phthisis) is as dangerous as it is painful (see following section). The pain, difficulty in swallowing, and other troubles are often so prominent as to throw those referred to the lungs into the shade.

In rarer cases, there is paralysis of the recurrens, which may be due to compression by enlarged tracheo-bronchial lymphatic glands (cheesy metamorphosis), or to the retraction of pleuritic adhesions. Brieger published a case of double paralysis of the recurrens, due to enlarged bronchial glands.

Inflammation and ulceration of the trachea are common, and may often be detected during life by tracheoscopy.

Betz states that goitre is found in youthful phthisical subjects.

Consumption rarely develops into gangrene of the lungs; Traube explains this by the infrequency of stagnation of the excretion in the air passages. If expectoration is checked, the sputum is liable presently to acquire a fetid smell, especially when the patient's strength is insufficient for vigorous expectoration, or the consciousness is obscured. Both the latter circumstances are unfavorable; fetid sputa in phthisis are therefore a bad sign.

Pleurisy is sometimes associated with consumption; it may be dry or moist, and in the latter case may cause a serous, or purulent, or hemorrhagic exudation. The exudation is susceptible of absorption, even in tuberculous pleurisy; though usually this improvement is only temporary, and fluctuates greatly. Fluid exudation sometimes checks the process of degeneration in the lungs, and therefore must not be regarded as entirely a bad symptom.

Pneumothorax in consumption is very much rarer than pleurisy (see Vol. I., p. 374).

I lately saw a case in which adhesion to the costal pleura preceded rupture of a cavity, so that subcutaneous emphysema covering a spot as large as a dollar occurred at the point of perforation.

The bronchial glands (as has been stated) sometimes cause paralysis of the recurrens by swelling and compression.

Abnormal dulness above the manubrium sterni sometimes enables us to recognize enlargement of the lymphatic glands. If cheesy glands become softened, they may rupture into a bronchus or the trachea, or if calcified, masses of calcareous material may be thrown out, simulating lung-stones.

Pericarditis is not frequent in phthisis; it may be tuberculous. In many cases, it seems to be excited by previous pleurisy; it rarely occurs

in consequence of rupture of a cavity into the pericardium. Cardiac thrombi are sometimes important as causing embolism of the pulmonary artery. Thrombosis of the pulmonary arteries and veins occurs, and the latter may cause embolism of the peripheral arteries, *e. g.*, the cerebral.

Digestive troubles are very common. Sometimes they are purely functional and unaccompanied by anatomical changes; for example, great want of appetite, or unconquerable dislike to certain articles of food. If this dislike is felt towards eggs, milk, meat, or other desirable things, the medical treatment is greatly interfered with.

Many patients suffer from troublesome eructations or obstinate vomiting; or profuse diarrhœa occurs, and continues for awhile, without any demonstrable changes in the intestines.

A feeling of heat, dryness, and burning pain in the mouth, especially on the tongue, is often encountered. The mucous membrane is of a peculiar bright red color, the papillæ of the tongue are swollen and prominent, the secretion of saliva lessened, and its reaction often sour; symptoms of catarrhal stomatitis. Superficial, partially aphthous ulceration may accompany it.

If the patient is very feeble, and his mouth is not kept carefully clean, oïdium albicans develops (Vol. II., p. 10). The tongue is covered with a thick, greasy-looking, yellowish-gray coat, easily recognized by the microscope as composed of roundish-longish fungus-spores and jointed mycelium. Excessive discharge of saliva often accompanies this, the fluid running from the open mouth almost continuously. The disease may extend to the throat and gullet, occasioning difficulty in swallowing.

Tuberculous ulcers are sometimes found on the tongue, said by Reverdin to have been first described by Ricord. There may be also a deep diffuse tuberculous-cheesy infiltration of the parenchyma, which may easily be mistaken for cancer of the tongue, or gummata.

Tuberculous ulceration or diffuse infiltration of the throat may occur. It is not usually recognized until there are slightly prominent yellow spots, irregular, often disseminated, and usually sharp-edged, and with indentations and angles. These cause very great trouble and pain in swallowing, though the subjective symptoms are often remarkably slight in even advanced cases. (See the following section on tuberculosis of the throat, for details.)

Do not confound the common prominent mucous follicles with gray transparent miliary tubercle, which is not very frequent.

The intestinal functions are often disturbed. At the beginning of the disease, constipation and diarrhœa alternate frequently. Catarrhal, tuberculous, or amyloid changes in the intestine, with extensive destruction of mucous membrane, often produce uncontrollable diarrhœa; pain may be absent, or may only be produced by pressure on the walls of the abdomen. In other cases, there are extraordinarily severe pains, which often take the form of attacks of colic. In a case of that kind in which I lately performed the autopsy, there were very extensive ulcerations, which in many places had approached close to the peritoneum. Shreds of intestine are sometimes plainly visible in the stool, and may be confounded with portions of undigested food. Obstinate constipation may occur, in spite of extensive changes in the mucous membrane. The best

proof of tuberculosis of the intestinal mucous coat is the presence of the bacilli in the stool, to be detected as before described. Compare a subsequent section upon intestinal tuberculosis.

Ulceration of the intestine may also cause hemorrhage which is apt to be abundant and hard to control. Dumas rightly says that hemorrhage is not very common, as the ulceration generally extends slowly, and gives the blood-vessels time to become obliterated; but very small ulcers have been known to cause a fatal result.

Granger-Stewart says that hemorrhage is very common in amyloid degeneration, even if there are no ulcerations.

Peritonitis may be caused by intestinal ulcers; it is especially dangerous when caused by perforation.

Rectal fistula, usually tuberculous in its origin, is another complication of consumption; the older physicians considered that it was not injurious, but acted as a kind of derivant, and gave warning of the danger of closing it by an operation.

The liver is often changed in form and consistency, and enlarged. The principal changes are congestion, the fatty and amyloid metamorphoses, or a combination of these. A fatty liver is doughy in consistency, blunt-edged, and often hard to reach by palpation (see Vol. II., p. 212). An amyloid liver is usually larger, harder, and more elastic and sharp-edged; it is considerably less common than fatty liver, and is usually associated with hard splenic tumor (amyloid spleen) and albuminuria (amyloid kidney).

Splenic tumor in consumptives may depend on congestion or amyloid change. Large disseminated tuberculous-caseous spots are sometimes found in enlarged spleen.

Caseo-tuberculous inflammation of the testis and epididymis forms hard, uneven, knobby masses. Virchow has remarked that consumptives who marry and indulge too much in sexual intercourse, often have acute tuberculosis of the testis and prostate. The sexual appetite is often quite strong in consumptives, and is retained even when the strength is greatly wasted.

Menstrual disturbances generally appear very early. The menses are scanty and irregular, and at last cease entirely. Tubercle of the mucous coat of the uterus, the Fallopian tubes, or ovaries, has no clinical importance.

Pregnancy is an unfavorable complication; the destruction of the lungs very often becomes rapid towards the close of the period, and still more so after delivery; or a chronic case becomes acute; or general miliary tuberculosis begins. Uterine diseases are not rare in consumption, and very great caution is needed in treating them; some distinguished gynecologists have seen such increase in the phthisical symptoms after such treatment that they strongly advise not to employ it at all.

The urine often contains albumin. If there are but traces, general cachexy may be the cause of its presence; diarrhœa is often attended with transitory slight albuminuria.

If the quantity increases, and fibrinous casts are also present in the urine, we may suspect parenchymatous nephritis if the urine is also scanty, dark-colored, and of very high specific gravity.

Albuminuria, in the presence of symptoms of amyloid changes of the

liver and spleen, may be referred to amyloid change of the kidney, but parenchymatous and amyloid changes are almost always combined in the kidney.

Albuminuria is often due to congested kidney, but very extensive changes in the lungs are required in order to produce it.

Purulent urine is sometimes found, originating in tuberculous disease of the urinary apparatus.

Nervous symptoms are almost always of serious import. Violent headache is often the first; referred either to the frontal or occipital region. Repeated vomiting often occurs after some time. If disturbed innervation of the iris muscles now appears (one pupil unnaturally large or small), the apprehension of tuberculous meningitis becomes almost a certainty. Stiff neck usually soon follows; consciousness is obscured, delirium occurs, and death follows, often immediately preceded by very great increase in temperature (hyperpyrexia).

These symptoms are not confined to tuberculous meningitis; the purulent form may occur, especially when caries of the temporal bones extends inwardly. The organ of hearing should be most carefully examined at the first appearance of meningitic symptoms.

Many patients complain of violent pains. The changes in the lungs do not cause pain, but pleuritic complications may; or there may be severe muscular pains, due to the exertion of violent coughing, or to parenchymatous changes in the muscles. They are either spontaneous or dependent upon pressure. Sometimes they are almost periodical, and occur (as I have repeatedly seen) at fixed hours in the afternoon.

Very severe muscular pains may simulate other diseases. I lately saw a man of thirty-five years who had been suffering violent pains in the muscles of the back and belly, whose physicians disputed whether to call it rheumatism or trichinosis. They had forgotten to examine the lungs, which contained cavities on both sides; profuse hæmoptysis occurred in a few days, and repetitions of it caused death in four weeks.

Continuous loss of sleep is often complained of, even when there is very little annoyance from cough and irritation. In my cases, this sign has usually been unfavorable, and has been soon followed by death.

Consumption is usually chronic, lasting many months or many years. The average duration of life in one thousand cases collected by Williams in his practice was seven and a half years. It is, therefore, easy to infer that cases differ widely among themselves.

The disease may be acute from the beginning, and last but a few weeks; such cases occur chiefly in young persons. The terms "phthisis florida" and "galloping consumption" have been applied to such cases. They usually involve extensive changes in the lungs, or a tendency to unusually rapid progress.

A chronic case may be interrupted by an acute stage, which often directly causes death; the exciting cause of which may be pneumonia, pleurisy, pericarditis, or peritonitis. General miliary tuberculosis often originates in chronic consumption of the lungs. The impulse to the acute form may be given by various injurious circumstances.

Von Buhl mentions purulent peribronchitis as the cause of a very rapid death.

Though death is the usual termination, yet cure, or decided improvement now and then occurs. If the tubercular parts of the lung heal completely, a contracting cicatrix of connective tissue remains. Imperfect cure is more frequent than perfect cure; there is a diminution of local diseased action, but we are not sure that it will not again become progessive.

If there is a slow and steady progress of the disease, the final symptoms may be those of debility. Profuse hemorrhage may cause death unexpectedly. Œdema of the glottis, in disease of the larynx, may be fatal. There may be such difficulty in swallowing as to produce death by inanition. Suffocation may result from very extensive disease of the lungs, or from compression by pneumothorax or pleuritic exudation. Death may occur with symptoms of general dropsy. Or, it may originate in the circulatory apparatus, through embolism or thrombosis of the pulmonary artery. Fatal embolism of the cerebral arteries, originating in thrombi within the pulmonary veins, has been observed. In a few cases, the cause of death was not demonstrated by the autopsy, and nervous disturbances might account for it (Perroud).

III. Anatomical Changes.—Either lung, or both, may be affected. The disease is often limited to the apices, while in other cases an entire upper lobe or a great part of it is involved, and in yet others the lower lobe is involved; but the process is apt to be most advanced in the upper ones. Cases in which the lower lobes are chiefly or exclusively affected are exceptional. The region around the hilus and the lower lobe are mentioned by Michael and Weigert as a favorite spot for the disease (the apices remaining untouched) in children only.

Among the characteristic macroscopic changes, caseous degeneration is prominent; a more certain sign is the microscopic evidence of the presence of the tubercle bacillus. The more recent the diseased portion is the more abundant are the bacilli; in caseous masses they diminish, though the spores seem to be retained, which must render the caseous mass contagious. It is very probable that the bacilli are spread by coughing out portions of contagious secretion, which is drawn back on inspiration into other parts of the lung. The cheesy masses in the lungs originate in a transformation of inflammatory products; the part chiefly involved in inflammation is the parenchyma; hence, consumption has by many been considered identical with caseous pneumonia.

The cheesy portions are easily recognized by their yellow color and crumbly, friable, pulpy consistency. A lobular arrangement is often seen, the lobules often separated from one another by tissue in a state of slaty induration. The smallest are less than a pin's head in size. In other places, several lobular spots unite to form large caseous surfaces, the lobular origin of which may often be traced by the irregular angular contours. Uniform diffuse caseous change of an entire lobe is very much rarer.

The bronchi that pass into a caseous part are frequently dilated. They often contain a secretion which is purulent, or thickened like cheese, or translucent like jelly. This can be forced out by pressure, but when in situ is easily confounded with miliary tubercle, especially as it projects somewhat on the cut surface. Ulcerative changes of the bronchial mucous membrane are often seen; or knotty thickening of the bronchial wall with connective tissue, partly caseous, especially on the outside (peribronchitis).

The cheesy product is not developed as such; it consists of degen-

erated inflammatory products which have suffered the fatty change and great loss of water at the same time. In the stage preceding, we find masses like frog's spawn, translucent-gelatinous, gray-transparent or gelatinous, formerly termed gelatinous inflammation of the lung.

The cheesy deposit may become chalky or fluid. In the former case, salts of lime are deposited, beginning at the centre of the mass, and gradually changing the whole to a stony-hard substance. This is a kind of cure, as the lump remains as an innocent foreign body for the rest of life. The tissue sometimes softens around it, and it is detached and expectorated. The cheesy mass may not take the form of a stone, but may condense, and attract deposits of lime salts, till it reaches the consistency of mortar; a thick capsule of connective tissue (due to chronic interstitial pneumonia) then incloses it, and renders it permanently harmless. Very small caseous deposits may become fluid, and afterwards be completely absorbed, leaving a permanent cicatrix of connective tissue.

Liquefaction in a large caseous mass generally leads to the formation of cavities (vomicæ). The puriform mass becomes corrosive, and eats into some bronchus or bronchi, is expectorated, and leaves a hollow in its place, the inner wall of which is at first irregular and eaten into folds and shreds. Several cavities often unite, forming very irregular caverns. Their general tendency is to spread peripherally, but interstitial pneumonia often counteracts this by throwing around them a capsule of connective tissue. When the contents are entirely liquefied and ejected, the wall loses its tufted appearance, and becomes a smooth surface, usually covered with a more or less thick, caseo-purulent, crumbly mass, which can be easily scraped off with a knife. The cavity may be much larger than a fist, and sometimes may include almost a whole lobe of the lung.

The blood-vessels resist this process a great while; they may often be seen of the size of a pack-thread, crossing the cavity, their walls thickened and their opening usually obliterated. Stout branches may project into the cavity, sometimes presenting aneurismal dilatations, which frequently cause profuse and obstinate hemorrhage.

Cavities may also originate in dilatation of bronchi, which may be very large, and are usually recognizable by the direct passage of the wall of the bronchus into that of the cavity, and by the internal wall being covered with ciliary epithelium.

Tubercles are very often seen in addition to caseous nodules. Two kinds of tubercle are to be distinguished—the local and the disseminated. The former is usually best seen near a moderate-sized caseous deposit; it forms gray, translucent knots of tubercle, radiating peripherally, chiefly following the course of lymphatics. Gray tubercles are often seen near ulcers of the bronchial mucous membrane. Disseminated tubercle is scattered in small lumps through the lung-tissue; it may be observed on the walls of caverns; it often affects a great number of other organs (general miliary tuberculosis).

The above description relates to the macroscopic appearances. There exists a great variety of opinion in regard to the histological origin of the disease. The study of the disease has been made difficult by the fact that advanced cases, with very various combinations of disease of the several tissue-elements, are the ones usually examined.

The first question is, whether the disease begins as an intra-alveolar or an inter-alveolar process, or whether it first attacks the minute bron-

chi. All these processes are usually found combined; but it may be right to assume that one only was the original one.

It is a common error to consider that all cases have one point of origin. Of two such excellent observers as Colberg and Rindfleisch, the former affirms chiefly the intra-alveolar origin, the latter the inter-alveolar; is it not possible that both are right, but with too one-sided a bias? The determination of the point of origin of inflammation may often depend on accidental causes—catarrh of the small bronchi, an intra-alveolar process, or (perhaps the rarest) inter-alveolar changes. The essential point in consumption is bacillar infection of inflammation products followed by the caseous change.

Analyzing the processes, we find in the bronchi both peribronchial changes and those affecting the mucous membrane. The former is sometimes peribronchitis fibrosa, characterized by fibrous thickening of the adventitious tissue. The caseous form is more characteristic, shown by cheesy, often lumpy and knotty thickenings, on the outer wall of fine bronchi, and not to be confounded with tuberculous formations. In the cavity of the bronchi, we often see thickened caseous masses, the cross section of which in small bronchi may be mistaken for tubercle (encysted tubercle of older authors).

The intra-alveolar changes are marked by the filling of the alveoli of the lungs with cheesy masses. Caseous change is usually preceded by active desquamation of alveolar epithelium, so that at the beginning we have to do with so-called desquamative pneumonia. True tubercle formation also occurs here.

As Rindfleisch showed, the points where the finest bronchi enter the acini are the chief seat of inter-alveolar disease.

The muscles of consumptives are usually very pale and small. Von Buhl stated that parenchymatous changes are not rare. E. Fraenkel has further described granular opacity, pigmentation, atrophy of the muscular fibres, and hypertrophic changes of the perimysium internum. The changes are probably due to marasmus, and vary greatly in different muscles. When affecting the larynx, eye, or diaphragm, they may account for many functional disturbances.

The heart is usually small and flabby; its muscle is pale, sometimes deep-brown, occasionally with yellow fatty spots. The right heart is often dilated and hypertrophied. Fatty change of the inner coat of the pulmonary artery is often observed.

The bronchial lymphatic glands are often enlarged, or caseous, with tubercle or lime concretions. Large cheesy masses of lymphatic glands are especially frequent in children (Michael and Weigert).

The liver and kidneys may show marks of venous congestion.

IV. Diagnosis.—It is extremely hard to recognize consumption in its earliest stages. We may consider every case of apex catarrh as serious, and treat it with great care, but it is going too far to infer consumption and an inevitable doom as necessary consequences. It is here that it is of great importance to demonstrate bacilli. And the same is true of hæmoptysis, which may often be the sign of established tuberculous disease of the lungs. We must not forget that there are patients who have no expectoration and consumptives whose sputa are free from the bacillus. In such cases, we must make especial use of the accounts given us of consumption in the family, scrofulous disease in youth, and great tendency to disease of the respiratory organs. Increased tempera-

ture at evening and loss of bodily weight are symptoms not to be undervalued. Night sweats are important.

The above points must be considered when we have to decide whether an existing affection of the larynx is caused by concealed phthisis, or whether chlorosis, or disease of the stomach or intestine, is connected with phthisis.

In doubtful cases, the sputa might be inoculated into rabbits as a test, but it would require several weeks at least before we could expect to see tuberculosis develop in the animal.

If consumption follows acute pneumonia, the signs of infiltration persist; but there are often cases in which complete absorption and cure of pneumonic infiltration occur after a long time, and contrary to expectation.

If there exist signs of infiltration or cavities, we have to make a differential diagnosis. In case of infiltration, pneumonic disease is to be thought of; consumption has not the cyclic course of pneumonia, and the fact that the apex is chiefly affected may often be decisive in favor of consumption; an affection of both apices is in favor of the latter.

Tuberculous cavities have not the stench of those caused by gangrene, or the characteristic three layers, or the mycotic bronchial plugs in the sputa. The same is true in the case of bronchiectasia with putrid destruction of tissue; this is also chiefly located in the posterior and lower parts. In consumption, we do not generally see the pieces of lung tissue in the sputa which we see in cases of abscess, nor the abundant mass of hæmatoidin crystals, which are almost always seen in the latter.

V. Prognosis.—This is very serious, and in the majority of cases unfavorable. Recovery is hardly to be expected unless the case is seen at the first stage, and unless the patient can make pecuniary and other sacrifices; there is hardly a disease in which so much depends on the purse. If one can leave home and live at certain spots, free from care and bodily exertion, life may often be protracted for many years; or in favorable cases complete cure may be obtained.

The age affects the prognosis, for in young persons the disease is apt to run a quick course, and is less disposed to recovery or improvement; we sometimes find consumptive parents of advanced years who lose one child after another by their own disease.

Hereditary predisposition, being beyond the reach of medicine, makes the prognosis more unfavorable.

External circumstances may make the prognosis very unfavorable; for the poor, who are forced to remain in bad lodgings, to eat insufficient food, to over-work themselves in dusty factory-rooms, can hardly hope for improvement and cure.

Marriage is unfavorable, for we often see the phthisical process increase and become acute very soon after marriage.

The existence of extensive disease is unfavorable, especially as such cases have a tendency to assume the "galloping" form.

The prognosis is serious if cavities are present.

Complications may add unfavorable features; some, as the amyloid change, can hardly admit of recovery.

The number of bacilli in the sputa has been found to bear no relation to the extent and probable course of the disease.

VI. Treatment.—Rational preventive measures are worth more than

all that physic can do for an established case. There is little enough, however, that a physician can do by way of prevention, nor will his warnings be of much avail until more energetic steps are taken by the State or by public associations. Among the chief requirements in this direction are healthy and well-lighted tenements for the working classes, State supervision of factories and factory-hours, and attention to providing abundant and nutritious food.

In individual cases, there is need of special care, dating from the first day of life, for children that spring from consumptive families or from parents advanced in years or exhausted by disease—such parents being often known to transmit a phthisical constitution. The mother must be forbidden to give suck, and a good wet-nurse must be provided, whose freedom from consumption must be ascertained by examination. If artificial food is given, let only thoroughly boiled milk, taken from one cow that is free from disease, be used. As the child grows older, let him be well fed, kept from overwork at school, hardened by the judicious use of cold-water frictions; let him practice such gymnastic drill as is fitted to develop the chest and lungs and strengthen the chest and muscles, and let the choice be made of an occupation which requires wholesome movement in the open air. All diseases of the respiratory organs in such persons require special care. We hardly need say how often these measures must remain theoretical, simply for want of means to carry them out.

Scrofulous disease must receive energetic and persevering treatment. All diseases of the respiratory tract must be treated with uncommon care, especially those which, like measles, scarlet fever, or whooping-cough, frequently lead to consumption.

Marriage requires serious consideration in persons who have consumption or are suspected of having it. They should be dissuaded from marriage; it will put them in danger of a more speedy development of the disease, or of hastening its progress; and inflicts a blight on the posterity.

An important rule of prophylaxis relates to intercourse with consumptives. There can hardly be a doubt that the disease is contagious, and we know that the contagious element is contained in the sputa. As protective measures, we should forbid the patient's sleeping with another person. The sputa must be kept in a covered glass and disinfected with a five-per-cent solution of carbolic acid, of which an amount is added about equal to the daily discharge. Fischer and Schill have proved that this destroys the tubercle-bacilli within twenty-four hours; but they also found that the bacilli retained their vitality for six months in dried sputa. The bare possibility of contagion makes such precautions necessary; nor is it a sufficient answer to point to this or that person, or to many persons, who have received no harm from such contact. We must also be careful in using the clothing or linen of such patients; and must disinfect it.

Consumptives treated in hospitals must be isolated, shut out from the general wards, and, in particular, not placed with those who have non-tuberculous diseases of the respiratory tract.

In treating developed cases, we must lay much less stress on medicines than on residence in a well-selected climate-cure. The "bath-cures" or spas are often visited merely as a matter of fashion; but in consumption the case is quite otherwise, as will be admitted by every physician who has treated many consumptives of means.

If climate-cures are to be of use, they must be employed as soon as possible, for we can expect no benefit from them in those who present large cavities or extensive infiltration. Patients with one foot already in the grave are often sent off at the risk of life, only to return speedily, with their false hope gone, seeking their home to die in it. In the early stages, it will be well for those who have no restriction upon their actions, to visit climate-cures, even if there is only a suspicion of disease, for it is better to do this too early, or needlessly, many times, than to do it too late once.

There is a very large number of climate-cures, and of late the number has much increased, often to the injury of the cause. We do not attempt to give a long list. It is necessary first to decide between the limited or "closed" establishments, like Görbersdorf in Silesia, Königstein in the Taunus, Inselbad near Paderborn, and Reiboldsgrün in Saxony, and the "open" health resorts. The former have been rather objected to since the discovery of Koch's bacillus; they certainly should only be visited by decidedly tuberculous patients, not by those merely suspected, owing to the danger of contagion; and they must be well aired and carefully disinfected. Among the latter we can highly recommend from our own experience for summer residence Kreuth in the Bavarian mountains, while we place the localities in the Hartz, as Andreasberg, in the second list. For autumn and spring, select places in Tyrol (Meran, Gries, Görz), in Upper Italy (Arco, Cadenabia, Lugano, Palanza), or on the lake of Geneva (Montreux, Clarens). For winter residence it is doubtful whether to prefer a high region with cold equable climate, or a southern place with mild climate. For patients from the northern parts of Europe we prefer high localities; Davos, with its excellent arrangements, gives extremely good results. Not a few patients of mine, who have spent several whole winters in southern places, have complained that after returning home, with all due precautions, they have found the change of temperature so disagreeable that the benefit they had derived soon disappeared. The residence at Davos, however, has agreed with them wonderfully, and the benefit has been permanent. Persons disposed to hæmoptysis and affected with laryngeal disease should not generally go to Davos, but to southern climates. In all cases, patients must stay as long as possible in the place selected, must visit it repeatedly, and not return home until settled warm weather is established.

Among the climate cures of the south we mention San Remo, Mentone, Monaco, Nervi, La Spezia, Cannes, Hyères, Pau; also Pisa, Florence, Venice, Rome, Palermo, Catania, Ajaccio, Malaga, Cairo, Algiers, Madeira, and Malta. We have known several very serious cases in which Madeira, especially, produced surprising results, but the symptoms very quickly returned as soon as the patient came home, and it was necessary to take up a permanent residence in Madeira.

In summer, much good may be done by residence in sheltered country places with good food and much exercise in the open air. Thüringen, Bavaria, Baden, and especially Switzerland, are very rich in summer resorts.

Residence on the sea coast has been again recommended of late; Wiedasch, among others, says that in Norderney consumption is very rare. Long sea voyages have been found useful; Maclaren, for example, advises them strongly, but Jones opposes them. The use of the grape cure and the whey cure is diminishing.

But a very small part of our patients will be able to make use of these remedies; as for the rest, treatment offers little for them.

Much care must be taken with the diet and habits; it has been said with reason that a phthisical person should consume more food than he really needs. It has even been recommended by many to introduce food by the stomach-tube in cases of unwillingness to eat.

Let the patient have in the morning several cups of milk, or cocoa, or coffee with egg. At the second breakfast, a soft-boiled egg, shaved ham, tender meat-sausage, white bread and butter, and a glass of port. At dinner, meat soup, good meat, well-stewed fruit, and half a bottle of good red wine. In the afternoon, milk or cocoa. At supper, porridge of wheat-grits or rye, soft egg, ham, cold meat, white bread and butter, a glass of good Bavarian beer. A record should be kept of the person's weight. He should go to bed from 9 to 10 P.M., and rise at 7 or 8 P.M. On calm days, a walk in the open air; but neither go out too early, nor return too late. All bodily and mental exertions are to be avoided.

Cod-liver oil is an important remedy, especially indicated for thin persons. Give from one to three tablespoonfuls in the morning; but leave it off when loss of appetite or diarrhœa occurs. Malt extract has also a reputation.

Inhalation of iodine, iodide of potassium taken internally, arsenic, and salts of phosphorus have been recommended as specifics in consumption, but we must say that there are no specifics known, and that our treatment is purely symptomatic.

Buchner's recent recommendation of arsenic has aroused attention, and both favorable and unfavorable reports have been given. We have repeatedly seen very good results at the beginning of the disease, but no specific results as against the lung changes themselves. Appetite was improved, weight increased, febrile movement ceased in several cases; after a time, even night sweats were lessened. In extreme cases with great cavities and extreme weakness, we have neither seen nor expected success from arsenic. We have found the combination of arsenic and creasote often useful, if the secretion was very abundant in the air passages, and expectoration difficult. (℞ Creasoti, gr. vi.; acidi arseniosi, gr. ss.; adde vehic. q. s. ut ft. pil. no. 20. Consperge cortice cinnamomi. S. One pill three times a day after eating.)

The transfusion of lamb's blood, and the use of benzoate of soda, may have some historic interest; Brachet recommended inoculation with small-pox, saying that he had seen phthisis of the lungs cured after that disease.

Steinbrück and Krull successfully employ inhalation of oxygen.

Surgical operations have been employed, but without brilliant success. Among the elder physicians, Von Herff and Hoken have advised the opening of cavities and surgical treatment of them; more lately, Mosler, Pepper, Mutchinson, and Williams have repeated the experiment, by puncture or incision. Koch advised the injection of irritating fluids (carbolic acid or tincture of iodine) into the lung-tissue, to excite cicatrization.

In every case, a record of weight and temperature must be kept, and must be continued a long time after cure seems to be attained. The spirometer shows the vital capacity of the lungs, the pneumatometer the pressure exercised in expiration and inspiration; both are much used as tests.

For violent cough, use small doses of narcotics, but be cautious with such remedies, as it is easy for a patient to acquire a habit of using them in the course of a chronic disease.

For abundant secretion in the bronchi use expectorants. In such cases, the use of the waters in Lippspringe, Weissenburg, Selters, Ems, Soden, Obersalzbrunn, Gleichenberg, etc., has been recommended.

If anæmia is very prominent, use mild preparations of iron. A mixture of iron and chalk is often advisable; *e. g.*, ℞ Ferri lactat., calcii phosph., āā ʒ iiss.; magnesia carb., sacchari albi, āā ʒiv. M. S. A knife-point three times a day after eating.

The iron waters of Pyrmont, Driburg, Cudowa, Reinerz, Salzbrunn, Konigsdorf-Jastrzemb, Steben, or Spaa may be useful.

For loss of appetite bitters are suitable, as quinine in small doses, $\frac{1}{6}$ gr. three times a day [? 1½ gr. Transl.]; compound tinct. cinchona bark (decoct. cinchonæ, 1 : 18, ℥ vi.; ac. hydrochlor, gr. xxx.; syr. simpl., ʒ v.; M. S. Tablespoonful every two hours); or folia trifolii fibrini (℞ Fol. trifol. fib., gr. xxiv., boil with equal parts water and claret wine and strain to make ℥ vi.; simple syrup, ʒ v. M. S. Tablespoonful every two hours).

A careful use of Carlsbad or Kissingen water is sometimes indicated.

For febrile symptoms, antipyrin is the surest remedy (ʒ i. in ℥ iss. of water by enema).

Heavy sweating is quite amenable to atropine (Wilson, Fraentzel, Williamson) (℞ Atrop. sulph., gr. $\frac{1}{100}$; pulv. althææ, q. s. ut. f. pil. no. x. S. One or two at night). In many, it causes diarrhœa, which obliges the patients to suspend its use. Seifert recommended agaricin (gr. $\frac{1}{16}$ to $\frac{1}{8}$) and Frærntzel, hyoscine (gr. $\frac{1}{120}$). Cauldwell has lately used with success picrotoxine (gr. $\frac{1}{4}$ for a dose). Köhnhorn recommends powdering the patient with salicylic acid (℞ Ac. salicyl., ʒij.; starch, ʒ iiss.; talc., ℥ iij., M.). If the skin is very dry, it is to be oiled before powdering. Lauder-Brunton has used strychnine successfully. The elder physicians used also sage tea, and boletus laricis. I have repeatedly seen good results from friction in the evening with cold water, to which a few spoonfuls of alcohol or cologne water had been added.

The treatment of hæmoptysis is given in Vol. I., p. 256. That of diarrhœa, in a following section on intestinal tuberculosis.

2. *Laryngeal Phthisis, Phthisis Laryngea.*

(*Chronic Ulcerous Tuberculosis of the Larynx*).

I. Etiology.—Laryngeal phthisis includes all those ulcerative processes in the larynx which are produced by the colonization and growth of tubercle bacilli, as in the lungs. The disease has a deservedly bad reputation, as it causes very painful symptoms which often make the patient's life a torture.

The disease is more common in men than in women; it is rare before puberty, and most frequent between 20 and 30 years of age.

As a rule, it is associated with tubercle of the lung, and in the majority of cases is secondary. It is suspected, not without reason, to be often due to self-infection by bacilli in sputa; such infection would be favored by accidental catarrh, straining of the vocal cords, and perhaps by a want of resisting power in the larynx. The lung disease is usually fully developed before signs of the laryngeal trouble appear, and sometimes it is almost the last symptom during the course of the disease. Yet cases are seen in which the larynx is very deeply involved before the first symptoms appear in the lungs.

Heinze found among 1,226 autopsies of consumption made at the Pathological Institute of Leipzig from 1867 to 1876, 376 cases of laryngeal phthisis, or 30.6 per cent. Mackenzie gives a similar result (33 per cent) from his own experience.

It is stated by good authorities that sometimes all disease of the lungs has been absent, making the ulceration of the larynx independent or primary. It is even said that the lungs have been the part secondarily affected. The existence of the primary form can only be proved by autopsies, which at present are wanting, for we cannot certainly affirm that the lungs are unaffected merely because physical signs are wanting. We personally believe in the existence of the primary disease, and think that the predisposition of lung-tissue to tuberculosis has been overrated. Sommerbrodt has experimentally shown that primary disease of the larynx may cause secondary changes in the lungs.

II. Anatomical Changes.—By Koch's revolutionary discovery, the domain of laryngeal phthisis has been sharply defined, its sign and stamp being the presence of the bacilli in the diseased parts.

As regards the histogenesis of the disease, later researches, especially those of Heinze, show that the mucous and submucous coats of the larynx are thickened in most cases, owing to the accumulation of round cells, and more especially to the development of numerous tubercle nodules. The mucous coat is pale, gelatinous, and its surface often irregularly nodular. The nodules become caseous, and afterwards break down, producing tuberculous ulcers of the mucous membrane.

The changes above described are not always limited to the larynx, but are often met with in the pharyngeal parts, the trachea, and sometimes the bronchi, showing, as it were, the route taken by the sputa. Sometimes the ulceration extends directly from the mucous membrane of the larynx to that of the fauces or trachea. Tuberculous ulcers in the trachea alone are rare.

There is much variation in the size and appearance of the ulcers. The size may be that of a pin's head, or a pea, or large parts of the surface may be covered. The shape is often circular; sometimes they are irregular. The loss of tissue may be superficial or may extend deeply; in the latter case, they often have the form of a funnel or crater, which is made more distinct by the wall-like thickening and prominence of the edges. Papillary excrescences of the mucous membrane or epithelial growths of the edge of the ulcer are sometimes found. The base of the ulcer often has a yellowish or tallowy-gray coat; it is more rarely clean and red, and a layer of pus is found only at the edge.

Some of these ulcers can be called follicular ulcers of the larynx; Rindfleisch has particularly studied their origin. They originate in ulceration of the outlets of the glands of the mucous membrane. A roundish and flat-funnel-shaped ulcer first appears, and afterwards spreads both downwards and laterally. The glandular body at the same time perishes under the same process, and the ulceration finally involves the perichondrium. If neighboring ulcers run together, they produce indented or (as Rindfleisch well says) bunch-of-grapes ulcerations.

Marked tuberculous tumors form in rare cases in the larynx or trachea (Chiara, Mackenzie).

A very favorite seat of these ulcers is the posterior wall of the larynx in the inter-arytenoid region. They are often formed on the mucous

membrane of the vocal processes and the posterior end of the true vocal cords, on the false vocal cords, the mucous membrane of the arytenoid cartilages, and the epiglottis.

If the ulcers extend, severe secondary alterations of the larynx may occur. The insertions of the muscles of the cords are often destroyed, or the cords themselves are separated from the vocal process of the arytenoid. Large parts of the larynx may perish completely, the chief part of the epiglottis especially being often quite destroyed. Perichondritis or œdema of the glottis with fatal results sometimes supervenes.

The changes may be limited to one side, or chiefly so; it is said that they often are confined to the side corresponding to the lung which is most affected.

The cartilages are often found ossified, even when the perichondrium is not implicated in the ulcerations.

Fauvel remarked the almost invariable freedom of the laryngeal (*i. e.*, cervical) lymphatic glands from secondary tuberculous changes.

It is a peculiarity of the tuberculous ulceration of the larynx that it has very little tendency to heal, and a great disposition to spread. Cicatrization is extremely rare.

III. Symptoms.—The laryngoscope is of leading importance in recognizing this disease. The changes begin with swelling, the surface of the larynx being often uneven as if warty, and pale; ulceration follows. The swelling of certain parts may be so great as to confuse the appearance of parts.

Many authors claim to have seen miliary tubercles with the laryngoscope, but this is perhaps not trustworthy. If ulcerations exist, we cannot always recognize them with the mirror, even when there is little swelling. An ulcer on the posterior wall of the larynx, in particular, may escape notice, or its upper edge alone may be seen. Very small ulcers may be overlooked. A deposit of pus and mucus may temporarily conceal an ulcer.

Impairment of the voice is a prominent feature, and may vary from slight hoarseness to complete aphonia. We often perceive a striking disproportion between the slight ulcerative changes and the severe affection of the voice, showing that ulceration is not always the sole cause. Swelling of the mucous membrane and paresis of the muscles of the cords may be concerned in producing this effect.

Tickling, or piercing pains in the region of the larynx are usually felt; the pain may become intense, and radiate towards the ears.

There is generally a very strong impulse to cough, not only felt by day, but destroying sleep by night. Muco-purulent or puriform masses are expectorated, which may be mixed with streaks of blood, and under the microscope sometimes show elastic fibres, which are less curled than those from the lung.

Trouble in swallowing is very common; fluids especially go the wrong way and provoke coughing, owing to incomplete closure of the glottis. The act of swallowing is sometimes so painful that all food is refused; this is especially the case when the epiglottis and false vocal cords or the arytenoid bodies are swollen and partly destroyed, as in such cases every morsel presses on the diseased parts.

The lungs usually show marked phthisical disease, but laryngeal phthisis sometimes seems to stand alone.

Death may be due to exhaustion with increasing hectic symptoms; or it is caused suddenly by œdema of the glottis, or is associated with the above-described symptoms of laryngeal perichondritis (see Vol. I., p. 189).

Cures are excessively rare. I have seen two cases, in both of which the disease continued in the lung, and death occurred in the space of one year and one year and a half respectively. The formation of contracting cicatrices is equally rare.

IV. Diagnosis.—With the laryngoscope, the diagnosis is easily made, for gelatinous thickening of the mucous membrane or ulcerative processes in the larynx in a case of consumption are almost decisive. To this add that we can remove secretion from the surface of the ulcer with a clean brush or sponge, and examine it for the tubercle bacillus. The latter point is chiefly of importance when there are no decided changes of the lung, and we desire to exclude the possibility of syphilitic ulcers. Yet a combination of the latter with phthisis of the larynx and lungs is not rare. An examination of sputa alone is not decisive, for the tubercle may come from the lungs; and in taking out the secretion from the surface of the ulcer we should be careful that we are not taking sputum that has stuck there.

V. Prognosis.—The nature of the disease renders the prognosis usually unfavorable. Recovery can hardly be expected. The disease usually runs a very speedy course to death, the longest period after the first laryngeal symptoms being usually a year and a half.

VI. Treatment.—We should avoid all energetic local measures. If there is severe pain, or trouble in swallowing, muriate of cocaine may be pencilled on the parts (solution of one part in twenty) several times a day; this often gives brilliant results, surpassing those of morphine given by insufflation, by pencilling, or by subcutaneous injection in the neck. Let all food be in the form of thick porridge, as far as may be, since that is less apt to go the wrong way.

If inclination to cough is strong, give narcotics and inhalations, for which we especially recommend solution of bromide of potash (gr. xxx.: ℥ vi. of water every three hours) or morphine.

If the sputum has a bad smell, use inhalations of carbolic acid (0.5 per cent) or liquor aluminii acetici.

Do not relax attention to the main disease; and treat complications according to known rules.

With many other physicians, I have seen no good results from touching the ulcers, or insufflation; sometimes there has been distinct harm. Nitrate of silver, iodoform (Lincoln), and bromide of ammonium (Gerhardt) have been recommended.

The two cases of recovery which we mentioned (demonstrated after death) resisted for many months all treatment by inhalation and touching, and did not begin to heal until every kind of treatment of the larynx had been given up for a considerable time.

Appendix.—Chronic tuberculous changes—usually tuberculous ulcers—may affect the mucous membrane of the nose as well as that of the larynx. They are commonly secondary, but are said to have been seen as primary.

3. *Phthisis of the Pharynx. Phthisis Pharyngea.*

(*Chronic Ulcerous Tuberculosis of the Pharynx.*)

I. Etiology.—Tuberculosis of the soft palate and throat may be primary or secondary. In the latter case, tubercle is developed second-

arily on the mucous membrane of the soft palate and throat of consumptive persons; in the former, the disease appears in otherwise healthy persons. It is not always easy to be sure that a case is primary, as consumption may lurk concealed. If pharyngeal phthisis appears during consumption, we may infer infection by the sputa. If the throat is the primary seat of disease, the cause may be eating of tuberculous food, or inhalation and retention of pulverized tuberculous sputa. We know nothing exact in regard to the mode of infection.

Most of the cases have been reported by French and German authors, and the patients have been all adults, with hardly an exception—Isambert's case of a girl aged four and a half not seeming free from dispute. I have been struck with the frequency with which persons have been affected who were previously syphilitic. My cases, and those of foreign authors, are chiefly of men.

II. Symptoms.—The disease often gives very little trouble. The feeling of dryness, scratching, and tickling in the throat and pain in swallowing are often very trifling, even when extensive ulceration of the mucous membrane exists; but some patients are tortured by very severe pains, which occur spontaneously or are provoked by the act of swallowing, and often shoot to the ear. The development of the process can be followed from the beginning in many cases. Pearl-gray, transparent nodules start up, which afterwards become yellow and cheesy, and break down, forming shallow ulcers, which spread, unite, and become deeper. In other cases, there is loss of substance of the mucous membrane; the edges are usually sharp, sinuous, raised like a wall, and fresh tubercles may be seen on or near them; there is much disposition to polypoid growths of the edges. The uvula is sometimes thickened, as if infiltrated with gelatinous matter. If tubercles are not found, but only ulceration, the distinction between tubercle and syphilis may be very hard to make, but the discovery of tubercle bacilli in the secretion of the ulcer is decisive. The loss of substance may be very great in the mucous membrane of the hard and soft palate and tonsils; in the latter Strassmann shows that tubercle is not uncommon. The cervical glands are usually enlarged and indurated.

The disease has not a fixed course. It may last as long as six months, but may be acute. Irregular high fever often exists. In a case of mine, general tuberculosis very soon appeared, and the patient died with symptoms of tuberculous meningitis. Death usually occurs through marasmus. Consumption of the lungs sometimes supervenes and carries off the patient; its relations to pulmonary phthisis are similar to those which it bears to laryngeal phthisis. In advanced cases, the larynx and intestine are often involved in the disease; or the gullet is attacked. The tongue often becomes tuberculous.

III. Prognosis.—The prognosis in secondary tuberculosis is similar to that of consumption; in the primary form, it is serious, but is not always unfavorable. Küstner, especially, has cured cases by local treatment. I lately saw tuberculous ulcers of the fauces heal, in a consumptive patient, while the lung disease continued to advance.

IV. Treatment.—In secondary tuberculosis, let the treatment be mainly symptomatic. Give chiefly fluid food; if there is pain, pencil the diseased spots several times a day with carbol-glycerin (1 : 25) or bromide of potash dissolved in glycerin (1 : 5), or if it is violent, use solution of cocaine (1 : 10). In primary tuberculosis, we ought to cauterize

early and actively, using nitrate of silver, chromic acid, or the galvano-cautery.

APPENDIX.—Chronic tuberculous change of the tongue has more surgical than medical interest; it comprises ulcerations or large tumors, easily confounded with cancer or gummata. The diagnosis is rendered certain by discovering tubercle bacilli in the products of destruction.

Tuberculous ulcers sometimes occur on the mucous membrane of the cheek and lips.

Tuberculous changes sometimes occur on the mucous membrane of the digestive tube, either spreading directly from the pharynx to the œsophagus, or occurring in the latter after a rupture of tuberculous tracheo-bronchial glands. They often cause no symptoms during life, and seldom lead to cicatricial stenosis (Beck and Chiari).

Tuberculous ulcers of the mucous membrane of the stomach are sometimes found at autopsies, but not usually unless there is extensive destruction in the intestine also. Litten has described a case in which there was one tuberculous ulcer in the stomach alone, which had caused no trouble during life.

Tuberculous ulcers of the stomach often do cause trouble; Paulicki observed perforation of the stomach; Oppolzer, fistula of the stomach, and Hattulo saw stenosis of the pylorus, which had been preceded by cicatrization and retraction.

4. *Intestinal Phthisis. Phthisis Enterica.*

(*Chronic Ulcerative Tuberculosis of the Intestine.*)

I. ETIOLOGY.—Tuberculous ulcers of the mucous membrane of the intestine are almost always secondary to consumption of the lungs. Cases of primary intestinal tuberculosis are so scarce that their existence is often denied, though, we think, incorrectly.

The association with lung disease is even more frequent than in the case of laryngeal phthisis. Heinze, for instance, found 630, in 1,226 cases of phthisis of the lungs (about fifty-one per cent), while only 376 (30.6 per cent) had signs of laryngeal tuberculosis. The ingestion of infectious sputa, or self-infection, is probably the usual method of production in cases of consumption, more especially as the gastric juice does not kill the bacilli. Intestinal tuberculosis is therefore a sort of food-poisoning. Such poisoning can occur in other ways, as by using the unboiled milk of tuberculous cows, or raw or rare-done meats from tuberculous animals. This is the simplest explanation for primary cases; it also explains the frequency of intestinal tuberculosis in children, almost exceeding that of lung-consumption; and is supported by Zippelius' statement that tuberculosis is especially frequent in countries where the pearly distemper is common among cattle.

II. ANATOMICAL CHANGES.—Tuberculous ulcers of the intestine agree with typhoid ulcers as regards the locality, being chiefly found in the lower part of the ileum, and the upper part of the colon. They vary greatly in number; sometimes there is a single one just below the ileo-cæcal valve, or ulcers in the vermicular process only, while in other cases they are very numerous, and extend over large segments of the intestine.

The phthisical changes always originate in the lymphatic follicles of the mucous membrane, either the solitary or the agminated. In the lat-

ter, the change usually affects a part, and not the whole mass of follicles. There is first hyperplasia of the cell-elements, so that the follicle enlarges, and projects as a little knot into the intestinal cavity. The new-formed cells compress each other, causing disturbance of nutrition; whence proceed drying, caseation, and breaking down, and afterwards softening and bursting, the result of which is the formation of a sharply defined, deep, crater-like ulcer—the so-called primary intestinal ulcer. If neighboring ulcers join, they form great patches of destruction—secondary ulcers of the intestine.

The lymphatic vessels which surround the blood-vessels of the mucous membrane in the form of loose sheaths are especially affected. The vessels originate at the mesenteric attachment, and pass around to the lymph-follicles on the opposite side; and the tuberculous ulcers, following a similar course, are apt to lie tranversely to the long axis of the intestine, forming belts or rings.

Through the medium of the lymphatics, tubercle is found underneath the serous coat. The ulcers are often found under the peritoneum, surrounded with a ring of nodules of tubercle; tubercles may be traced along the lymphatics for a long distance, beneath the serous coat.

The ulcers can usually be felt without opening the intestine; their edges can be felt as hard spots. The neighborhood of such places is often much reddened; the serous coat above them is thickened, opaque, and sometimes covered with fibrinous new membrane.

Attempts at cicatrization occur, but usually fail, for while an ulcer is healing at one part it is spreading at another. Yet cicatricial flexions and stenoses of the intestine are observed.

The mesenteric glands are usually involved. They are infected with tubercle bacilli, swell and become cheesy, and may soften. They are sometimes so large and numerous as to be felt through the walls of the abdomen as knobby lumps.

Koch has shown that tubercle bacilli take a prominent part in the origin of intestinal phthisis. The fresher the nodules of tubercle are, the more bacilli do they contain. The same is true of the tuberculous gland of the mesentery. The bacilli are remarkably abundant in intestinal tuberculosis.

III. Symptoms and Diagnosis.—The symptoms are suprisingly varied. Extensive ulcers of the intestines often remain undiscovered during life, if the examination of the fæces has been neglected. The process of examination is the same as in the case of sputa, and its value in diagnosis is the same—in spite of the possibility that bacilli may be swallowed and mixed with the fæces.

In other cases, symptoms of sudden peritonitis, perityphlitis and paratyphlitis, intestinal perforation or hemorrhage, hæmatemesis, or signs of internal bleeding, suggest the presence of latent ulcers. Perforation may occur spontaneously, or may be caused by the lifting of a heavy weight, straining at stool, playing on wind instruments, a fall, a blow, or similar occurrences.

We can scarcely name a symptom of intestinal ulceration which is pathognomonic of the presence of tuberculous bacilli. The stools are of chief importance. Diarrhœa is frequent, and is explained by the irritant action of the fæces upon the surface of the ulcers, which increases peristaltic action; to which add the fact that the absorbent surface is diminished by loss of substance. Catarrh of the mucous membrane will increase the tendency to diarrhœa. Several thin stools are often passed

in the early hours (2 to 5 A.M.), which has given rise to the expression "diarrhœa nocturna." In many cases the stools are unaffected, or various degrees of constipation exist. Such is the case when the small intestine alone is affected, especially its upper part, as this gives opportunity enough for condensation of fæces in the large intestine. Destruction of the nerves in the ulcers may annihilate reflex action, and so prevent diarrhœa. The muscular coat may be altered, and may be extremely slow in its reactions and functions. Obstinate diarrhœa sometimes alternates with periods of obstinate constipation. The stools in diarrhœa are thin, often smell like carrion, and sometimes contain undigested pieces of food, as potatoes, vegetables, tendon, and flesh, a condition termed lientery. Blood, pus, and fragments of tissue are especially to be looked for. Bloody stools show the presence of considerable amounts of blood, for small amounts are dissolved by the digestive fluids, and become unrecognizable. The microscope will discover blood much oftener than the naked eye. But the presence of blood does not allow us to infer an ulcer of the intestine until we can exclude other sources of hemorrhage.

Undue weight has been laid, especially by Nothnagel, on the occurrence of pus in the stool. It is easily understood that pus often is absent when there are ulcers, since pus-corpuscles can easily be dissolved and destroyed by the digestive juices. But in this respect we must not rely on gross appearances alone, for a cloudy appearance of masses of mucus found in stools often indicates the presence of numerous epithelium cells in a more or less altered state, instead of the suspected pus-corpuscles. Pieces of parenchyma from the mucous membrane will be found less frequently than pus.

Pain is a frequent, but not a constant symptom. It is sometimes extreme, and is described as cutting or boring. Sometimes there is no spontaneous pain, but it may be produced by pressure on the abdomen. Localized tenderness in the right iliac region is especially important, since ulcers are sometimes confined to the neighborhood of the valvula Bauhini.

Vomiting is rare, unless peritonitis occurs.

The appetite is generally poor, but cases of excessive hunger occur, especially in tabes mesenterica.

Indurated portions of intestine or enlarged mesentric glands may sometimes be felt through the walls of the abdomen, but fæcal masses must not be mistaken for them. The result is always fatal, due to increasing marasmus or the above complications (perforation, bleeding, peritonitis).

IV. Prognosis and Treatment.—The prognosis is bad; owing in secondary cases to the nature of the primary affection, and in primary cases to the want of tendency to healing.

Prophylaxis is important. The patient is to be warned against swallowing his sputa, and may try to disinfect such as he may swallow by stiff doses of alcohol, which is also useful for keeping up the strength.

If tuberculous ulcers are formed, give easily digested food, such as makes a small amount of fæces: milk, eggs, meat soup, solution of meat, and meat, but no vegetables, and little bread. If diarrhœa is present, give the astringents mentioned on p. 110, vol. II.

Opium, Dover's powder, columbo, cascarilla bark, and subnitrate of bismuth, are especially recommended. Injections of nitrate of silver (gr. iss. to viiss.), or injections of starch with opium, are useful. Sub-

cutaneous injections of morphine have lately been recommended (hydrochlorate of morphia, gr. xv.; glycerin, distilled water, āā fl. ℥ ss. M. S. ¼–½ syringeful).

We often have to combat constipation; solid masses may easily irritate the ulcer to a dangerous extent.

Use gentle laxatives; perhaps the best are those given in Vol. II., p. 122.

Pains must be relieved by warm cataplasms to the abdomen, subcutaneous injections of morphia, and chloral hydrate.

Complications to be treated as usual.

APPENDIX.—With tuberculous ulcerous changes of the intestine we must associate many cases of rectal fistulæ, originating primarily or secondarily, in connection with phthisis; they belong to surgery.

5. *Chronic Ulcerative Tuberculosis of the Urinary Organs.*

(*Renal Phthisis. Phthisis renalis, sive Nephrophthisis.*)

I. ETIOLOGY.—Tuberculosis of the urinary organs may lead to destruction of tissues and is always due to the introduction of the tubercle bacillus, which mixes with the remains of destroyed tissue in the urine, and is found in the sediment by the method described in Vol. IV., p. 276.

Chronic tuberculosis, as elsewhere, affects the urinary organs either primarily (*i. e.* independently and alone) or secondarily. In the latter case, the primary disease may be found in adjacent organs, as the sexual parts, or in the lungs. In the former case, we speak of a tuberculosis of the urino-genital apparatus. The secondary disease of the urinary organs usually arises from cheesy tuberculous changes of the epididymis, seminal vesicles, or prostate.

It is by no means easy to distinguish primary from secondary tuberculosis of the urinary organs with any certainty. Not only is it impossible to detect a very slight affection of the lungs, which may have been the cause of the disease in question, but a primary affection of the urinary organs may also have been the cause of the lung affection.

Chronic tuberculosis of the urinary organs is not rare; many cases have been hitherto regarded as chronic catarrh of the bladder or of the pelvis of the kidney. It appears most frequently between the fifteenth and the fortieth year of life, but has been seen after the seventieth year and before the completion of the third year. Men are affected oftener than women.

As for the path traversed by the infection, there is probably no doubt that when the lungs are the point of origin, the blood-vessels and lymphatics are the agents of transfer, as the bacilli may very easily enter them. This may be true even when the starting-point is in the genitals. Gonorrhœa with epididymitis is a frequent source of tuberculosis of the epididymis. The usual process is that the acute symptoms of inflammation of the epididymis disappear, but that a lumpy hardness remains, which subsequently receives bacillary infection, undergoes cheesy metamorphosis, and becomes a point for the further spread of disease. Concubitus also brings danger, for every well person who lies with one suffering from tuberculosis of the urino-genital apparatus is in danger of infection with tuberculous masses, either mingled with the secretions, or stagnating, in the form of tuberculous urinary sediment, in various places.

The causes of primary tuberculosis of the urinary organs are little known. Simple catching cold cannot cause it, for catarrh produces no bacilli; the most that it can do is to make the tissues hyperæmic and less able to resist the settlement of tubercle bacilli.

II. Anatomical Changes.—The anatomical changes in chronic tuberculosis of the urinary organs may affect nearly their whole extent, or may be distributed in spots, separated by intervals of sound tissue. A distinction may be drawn between ascending and descending disease, according as the peripheral organs or the kidneys form the starting-point. Many authors doubt the existence of the ascending form.

The kidneys are oftener affected on one side than on both; Meckel thinks the right kidney is more frequently affected than the other. The non-tuberculous kidney may be perfectly sound, or may be in a state of chronic parenchymatous nephritis; Badt and Rosenstein have described an obervation in which there was medullary cancer. Chronic tuberculosis of the urinary organs is rarely limited to the kidneys.

The alterations are characterized by cheesy infiltrations, at first of the papillæ, then of the entire pyramid, and at last of larger or smaller sections of the cortex. At the periphery we find gray tubercles, partially caseous, indicating the original point of infiltration. The caseous masses soften, break down, are expelled and carried off with the urine, and thus large cavities are produced in the kidney. There is therefore a true consumption of the kidney. The papillæ are the first to disappear, then the entire medullary cone, so that a section of the kidney shows the pyramidal portion replaced by irregular cavities lined with a layer of a cheesy, friable substance. The cavities are separated from one another by the calyces, but their lateral walls are often perforated or destroyed.

The process of destruction gradually extends toward the cortex. Numerous interstitial growths of connective tissue are here developed, but destruction of the cortex continues. At last there remains a large sac, formed of the connective tissue of the kidneys, but only in rare cases studded with miliary tubercles. In other cases, the sac embraces shapeless caseous masses, which sometimes have the consistency of brain (encephaloid).

The altered kidneys have generally increased a good deal in size and weight. In a case described by Klebs, the right kidney weighed 4 lbs. 13 oz. (normal weight 5¼ oz.), was 11¼ in. long, and 12¾ in. in circumference.

The pelvis is usually dilated, as detached caseous masses occasionally clog the ureters and dam up the urine. It is probable that tuberculous disease of the ureters often lessens the muscular power of expelling the urine. Tuberculous ulcers of the mucous membrane of the pelves, a cheesy friable sediment, and cheesy tuberculous infiltration of the submucous tissue are almost always found.

The ureters are often changed to rigid, knotty, irregularly dilated tubes, marked by the same changes as the pelves of the kidneys.

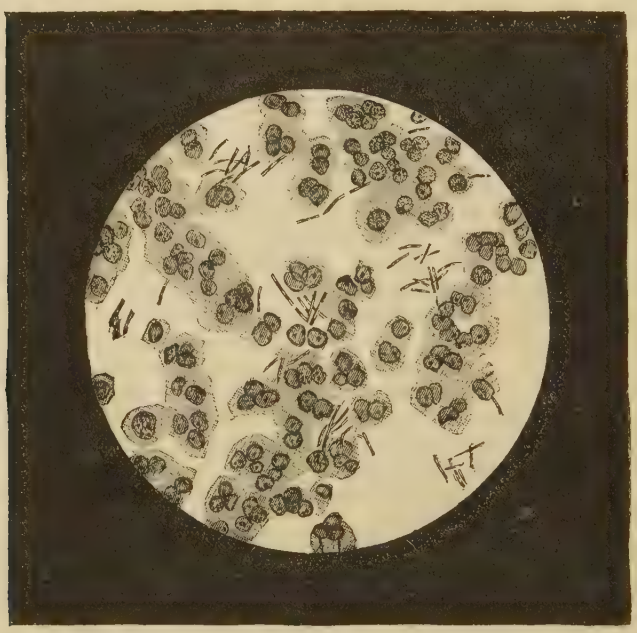
In the bladder, the first changes are usually developed at the fundus, near the neck. The cheesy tubercle breaking down gives rise to sharp-edged, sinuous-outlined ulcers with raised borders, at first often no larger than a lentil, but afterwards running together and thus gaining in size. They do not usually go below the mucous coat. The base of the ulcers is sometimes incrusted with phosphates.

Tuberculous ulceration of the urethra not infrequently causes peri-urethral changes.

Rupture has often occurred from the kidneys, the pelves, or the bladder, into the neighboring parts, such as the paranephritic connective tissue, the intestine, etc. Chronic tuberculous cheesy inflammations are often observed in the neighborhood of the organs affected with tubercle.

III. Symptoms.—These often hardly differ from those of a vesical catarrh: frequent desire to urinate, cloudy purulent urine, sometimes decomposed urine with ammoniacal or putrid odor, or that of sulphuretted hydrogen. We cannot be sure that chronic tuberculosis exists until we demonstrate the presence of specific tuberculous properties in the

Fig. 57.



Tubercle bacilli with spores from the urinary sediment, in chronic tuberculosis of the urinary organs, from a woman aged 30. Fuchsine and malachite green preparation. Immersion. 750 diameters. (Author's observation: Zurich clinic.)

purulent sediment. I have often succeeded in doing this by placing the sediment on a glass slide, distributing the drop finely by revolving the cover glass, shaking off the superfluous part, drawing the covering-glass several times through the flame, and then staining according to the rule given on page 276 of Vol. IV. Such a preparation is given in Fig. 57, containing abundance of tubercle bacilli with spores. But I must add that I have seen not a few cases in which, in spite of all care, I could not make the bacilli appear in the sediment, though the experiment was tried repeatedly and the diagnosis seemed established. There would then remain the inoculation-test, which is made by putting some of the urinary sediment, with antiseptic precautions, into the anterior

chamber of the eye of a rabbit. If the sediment contains specific germs, miliary tuberculosis will develop in about three weeks, at first in the iris, then on the other coats of the eye (Damsch and Ebstein).

The urine is usually light yellow, and very often increased in amount. Its specific gravity is usually unchanged. It often contains a very abundant purulent sediment, in which cheesy crumbs are often found, sometimes larger in size than a pin's head, the occurrence of which is almost characteristic of tubercle. Sometimes, along with wrinkled round-cells, free nuclei, and granular, sometimes fatty detritus, we find in these small bodies elastic fibres and components of connective tissue; and above all tubercle bacilli. We also find in the sediment round cells,

FIG. 58.

Urinary sediment in chronic tuberculosis of the urinary organs. The same patient as in Fig. 57. 250 diameters.

much detritus, epithelium cells from the urinary passages, and triple phosphates (comp. Fig. 58); the latter are often found even when the urine still has an acid reaction. The urine usually contains as much albumin as corresponds to the pus in it, and contains more only when chronic nephritis is added to the tuberculosis. Hæmaturia sometimes occurs.

If the bladder is implicated, the urine sometimes undergoes ammoniacal decomposition.

Desire to urinate, pricking and itching of the glans and meatus, are often complained of; and when large caseous masses pass out, there may be symptoms of stoppage of the passage. In some cases, vesical symptoms appear while the bladder is yet intact.

The matter must always seem suspicious when there are hard

(caseous) lumps in the epididymis, or in the prostate when examined per rectum, together with frequent micturition and purulent urine.

The implication of the kidneys can only be inferred when there are local renal symptoms. These include pain in the region of one kidney, which is sometimes very severe, and may extend to the back, towards the navel or testicle, and even to the thigh. Numbness is sometimes felt in the latter. The pain may be continuous or paroxysmal. Pain, however, is not a constant symptom, and sometimes it can only be provoked by pressure upon the region of the kidney.

A very important fact is the demonstration of enlargement of the kidney by palpation. The surface is usually smooth and sensitive to pressure. We sometimes succeed in observing alternate enlargement and diminution of the tumor. With enlargement, there are usually very severe pains and remarkably clear and scanty urine, while, when the urine becomes freer and cloudy, the pain diminishes. This is plainly due to temporary stoppage of the ureters by the detachment of large cheesy morsels, and hydronephrosis with retention of the purulent urine. I have repeatedly seen chills and high fever at the time of these occurrences.

In the case of a girl aged fourteen, I was enabled to diagnosticate tubercle of the ureter by the fact that a thickened, knotty cord was felt along the posterior wall of the bladder on the right side, by palpation per rectum.

Similar changes may sometimes be seen in chronic tuberculosis of the bladder.

In case of rupture outwardly, symptoms of paranephritic abscess, external renal fistula, vesical, rectal, or vesico-vaginal fistula, or periurethral abscess may occur.

The lungs are often tuberculous; and the same disease in the larynx and intestine may cause profuse diarrhœa, hoarseness, and trouble in swallowing. Sweats, chills, fever—in short, hectic symptoms—occur, strength is lost continually, and the result is death. The usual average duration is said to be one year, but cases lasting from ten to seventeen years are known.

IV. Diagnosis.—This is based on the demonstration of tubercle bacilli in the sediment of the urine, on the success of inoculation with the sediment, the occurrence of cheesy fragments in the sediment, the demonstration of local changes in the kidneys or urinary passages, and the existence of cheesy spots in the epididymis or prostate, or chronic tuberculosis of the lungs.

V. Prognosis—unfavorable, for treatment is powerless. But I have lately seen two cases in the Zurich clinic which recovered—both cases of primary tuberculosis. Persons with cheesy tubercle of the epididymis or prostate should avoid coitus, in order not to infect others.

VI. Treatment.—Treat the symptoms—give iron, quinia, and cod-liver oil to improve nutrition; if there is ammoniacal decomposition of the urine, wash out the bladder with disinfectants; send the patient to a good country place. Attentive care of gonorrhœa may be of prophylactic use.

Appendix.—Chronic tuberculosis of the male or females exual organs is not here described, being chiefly of surgical interest.

6. *Solitary Tuberculosis of Internal Viscera.*

Solitary tubercles are tuberculo-caseous foci which have run together and formed one cheesy tumor of varying size. An organ often contains only one large tubercle-focus, a solitary tubercle in the stricter sense; in other cases, several of them are found. They may grow to the size of a fist and larger.

The disease is almost always secondary, excited by tuberculous disease in other organs, chiefly the lungs. The symptoms may be prominently those caused by a growing tumor. The tumor may soften and break; or miliary tuberculosis may occur. We we will merely give a few special examples.

a. Solitary tubercle of the brain is most common in childhood. Its favorite seat is the gray substance, especially that of the cerebellum, where it frequently begins in the boundary between gray and white matter. It may grow to the size of a good-sized apple, but often forms only a knot of the size of a pea or a hazel-nut. In most cases, there is only one nodule; sometimes there are as many as twenty or more in different parts of the brain. Their form is roundish; more rarely irregularly knobby. The mass of the tubercle is dry, yellow, and cheesy, while its periphery often forms a gray translucent border. The mass is sometimes encysted, with distinct layers in the outer parts of the tubercle. Small nodules, becoming cheesy, are sometimes seen in the peripheral parts, showing that the large lumps were formed by a gradual coalescence of the smaller ones. If the tubercle extends close to the meninges, it may adhere to them.

Other metamorphoses occur besides the caseous, especially puriform liquefaction and calcification. Sometimes one-half liquefies, while the other calcifies.

The microscopic structure is like that of other tubercles. The chief mass is composed of round cells, giant cells also occur, and tubercle bacilli, especially in the latter. In the peripheral layers, fibrous tissue is more prominent, and many cases are distinguished by a striking development of fibrous substance—the so-called fibrous tubercle.

All cheesy substances in the brain are not tubercle, for the same change occurs in abscess, sarcoma, cancer, and especially gumma; the differential diagnosis of the latter would often be very hard if it were not for the presence of tubercle bacilli.

Primary tubercles (or such as are limited to the brain) are said to occur.

Symptoms may be absent; or there may be the symptoms of cerebral tumor (see Vol. III., p. 241). Cerebral tubercle may give rise to tuberculous meningitis or general miliary tuberculosis, after a previous latent course.

b. Solitary tubercles in the cord are said by Hayem to be most common in the lumbar enlargment, and to occur in children also. They sometimes reach the size of a hazel-nut; they cause no symptoms, or those of a tumor of the cord (see Vol. III., p. 98). They may exist simultaneously with solitary tubercle of the brain. In general, that which was said of the one applies to the other.

c. Solitary tubercles in the spleen are comparatively frequent in children that suffer from intestinal consumption or scrofulosis. Sometimes they are so numerous that the proper tissue of the spleen has almost disappeared. The organ may increase considerably in size, and have a nodular surface, making it possible to infer the diagnosis in view of the causation.

d. Solitary tubercles in the liver are rare; Orth has a good example.

e. Solitary tubercles in the muscle of the heart occur, but have only an anatomical interest.

7. *General Miliary Tuberculosis. Tuberculosis Miliaris Disseminata sive Universalis.*

I. Etiology.—In this form, the name tubercle, or little nodule (tuberculum), has its full anatomical significance, for the disease is characterized by the appearance of little nodules in many organs—which are gray, and translucent, and in the older stages opaque and cheesy-yellowish.

Before Koch's discovery of the bacillus, it was generally assumed that the process was an infectious one, but the knowledge of the poisonous element was derived from his studies. The chronic ulcerous tuber-

culosis of the different organs, and general miliary tuberculosis, have one and the same virus, in spite of the variety of their symptoms and anatomical changes.

General miliary tuberculosis is distinctly a metastatic disease.

The virus—that is, the bacilli—pass from any centre where they exist into the venous, lymphatic, and more rarely the arterial circulation, are carried in this way to all the organs, and, producing bacterial embolism, cause an outbreak of miliary tubercles. Hence it appears that general miliary tuberculosis is, in the majority of cases, a secondary affection. Whether the bacilli enter the general circulation directly from outside and at once produce general infection—that is, whether primary miliary tuberculosis exists—is still doubtful. Von Buhl was unable to find a primary source of infection in 10 out of 300 observations, but Simmonds lately found such a source in every one of 100 cases.

The disease most frequently associated is tubercle of the lung; Litten found it present in twenty-eight out of fifty-two cases, or fifty-four per cent. The same is observed next in frequency in tuberculous caseation of lymphatic glands, especially the tracheo-bronchial; but also after tuberculous psoas abscess, and tuberculous paranephritis and paratyphlitis. Tuberculous disease of the bones and joints sometimes causes miliary tuberculosis; Doutrelepont has described it as following lupus, which is nothing else than chronic tuberculosis of the skin. Tubercular pleuritis, pericarditis, and peritonitis are among the commoner sources of infection. In general, any sort of tuberculosis may become the starting-point of general tuberculosis.

Von Buhl has made the excellent observation that cheesy tuberculous foci are less likely to produce general infection in proportion as they are cut off from the general circulation by a thick capsule of connective tissue.

Sudden outbreaks of miliary tuberculosis are unexplained in many cases. The weather may be connected with it; cases are sometimes seen in groups, like epidemics, and Von Buhl states in regard to Munich, and Lebert in regard to Zurich, that cases are especially common in April and May. Other direct causes are loss of fluids (as in childbed), or care, sorrow, mental excitement. Injuries are sometimes given as causes; miliary tuberculosis is often said to proceed from operations on tuberculous bones and joints, especially when the operation is not wholly limited to sound tissue; and similar results are stated to have followed the removal of tuberculous rectal fistula. Litten states that general tuberculosis not uncommonly occurs after very rapid absorption of pleuritic exudations.

Reich has described a very curious instance of infection. Neuenburg, a town of 1,300 souls, divided its midwifery practice equally between two women, one of whom had consumption, and was accustomed to apply her mouth to the mouths of new-born infants to suck out mucus or to blow air in. Within two years ten children from this woman's practice died of miliary tubercle of the meninges, while none of those delivered by the other midwife had miliary tubercle. There was no inheritance of disease on the part of those which died; and meningeal tuberculosis was found to be a cause of death only twice in ninety-two children that died within the first year of life in Neuenburg.

Miliary tuberculosis does not exclude other diseases. It may coexist with typhoid fever (cases by Lavaran and Burkart), cancer (case by

Simmonds, cancer and miliary tubercle of the liver), and alveolar emphysema of the lung.

II. Anatomical Changes.—General miliary tuberculosis often infects most of the organs in one patient. In other cases, it is limited to single organs. As a rule, the salivary glands and pancreas are the only organs unaffected, but Barlow has described a case affecting the pancreas.

Simmonds gives the following table representing the percentage of frequency with which the different organs are attacked, based on one hundred cases:

Lungs	76 per cent	Peritoneum	26 per cent
Pleura	25 "	Pia mater	28 "
Pericardium	4 "	Dura mater	23 "
Liver	82 "	Brain	10 "
Kidney	62 "	Suprarenal bodies	2 "
Spleen	56 "	Thyroid	3 "
Intestine	57 "	Female genitals	2 "
Stomach	1 "	Striped muscles	2 "

Tubercle in the heart-muscle is also reported; in the endocardium (by Weigert); the inner coat of vessels (by Mügge and Weigert); in the thoracic duct, tongue, and fauces, marrow of bones (thirty-one per cent of Litten's cases); the choroid, retina, and iris.

In general miliary tuberculosis, as in many other infectious diseases, it is usual to find marked rigor mortis, ham-like discoloration of muscle, and granular cloudiness and swelling of the muscles and parenchyma-cells of internal organs.

Miliary tubercle of the lungs is easily recognized by the eye. The lung is everywhere filled with little gray transparent nodules, some so small as to be hardly visible, others as large as a poppy-seed, pin-head, and larger. The smallest are perfectly transparent, but the larger ones often have an opaque centre, or even the commencement of caseous change at the centre. The lungs feel full of little lumps when handled before cutting; sometimes reminding one of a bag of shot. The lumpiness appears on the cut surface also, and may be found more distinct by oblique light. The nodules can be removed with the knife-point.

In a few cases, one lung, or even but one lobe, is affected; but usually both sides are involved.

We usually note, in addition, symptoms of consumptive disease; retraction; frequently emphysematous change; recent pneumonia; œdema of the lungs; bronchial catarrh.

Miliary tubercle of the pleura is probably always associated with that of the lung; inflammation readily sets in, causing tuberculous pleurisy.

In the heart, the disease may affect the pericardial sac, the muscle, or the endocardium. Of the pericardium we may repeat what was said of the pleura. Miliary tubercle in the heart is most frequently seen beneath the endocardium of the right ventricle.

Miliary tubercles may be seen on the tunica adventitia, as well as on the intima of the blood-vessels; most commonly on the veins. Miliary tubercle of the thoracic duct was first described by Ponfick.

The spleen is usually enlarged, and often full of countless nodules, many of which are cheesy in the centre. If few in number and transparent gray, they may easily be confounded with the Malpighian bodies,

but they are more prominent and can be raised with the knife-point without damage, which we cannot do to the Malpighian bodies. In doubtful cases, the microscope decides, showing in the Malpighian body a peculiar central blood-vessel, and in the tubercle, bacilli. The capsule of the spleen may also contain miliary tubercles, often associated with inflammatory changes.

In the kidney, miliary tubercles are chiefly found in the cortex; while in the medulla they may be wholly wanting. They often form rows like strings of beads. They are most easily recognized on the surface of the kidney, where they distinctly project above the smooth surface. They are very often surrounded by a little ring of injected vessels. They are sometimes distributed along the ramifications of one branch of the renal artery.

The peritoneum is an extremely common site; especially the greater omentum. Peritonitis or ascites is very frequent, as a consequence of the tubercular deposit. The greater omentum is often thickened, dense, rolled up, and wrinkled.

Miliary tubercle in the mucous membrane of the stomach is rare. Strassmann and Chiari have discovered it in the tonsils and thyroid body.

The liver is often affected; the tubercles are found sometimes in the interlobular connective tissue, sometimes in the interior of the lobules, and form gray transparent nodules, or yellowish opaque bodies with indistinct boundaries. Increase of the interlobular connective tissue is a very frequent accompaniment.

The meninges, especially the soft ones, are often the seat of extensive miliary tuberculosis or tuberculous meningitis.

The bacilli are always found with the microscope in fresh miliary tubercle, and more especially in the giant cells, which are numerous. They become infrequent in proportion to the extent to which decay of the cells, necrosis, and caseous change occur; probably the spores alone remain in the cheesy detritus, and impart to it infectious properties. Koch has repeatedly shown the bacilli within blood-vessels.

An exact histogenesis of miliary tubercle cannot be given here; it has long been noticed that it first appears in the neighborhood of blood-vessels and lymphatics, and, according to trustworthy authors, often begins in the adventitial lymph-sheath.

General miliary tuberculosis has been a subject for many experimental researches, owing to the ease with which it may be produced in many animals by infection with cheesy tubercle. The first thorough experiments were by Villemin (1865).

III. Symptoms.—General and local symptoms must be carefully separated; the former being the consequence of general infection, the latter depending on the predominant affection of certain organs.

There is usually considerable fever; non-febrile cases are rare, but Lange mentions one. The type is variable, being sometimes continuous, sometimes remittent, sometimes intermittent. Brünniche has shown that an inverted type is common, with exacerbations in the morning and remissions in the evening.

The pulse deserves great attention; contrary to what occurs in typhoid fever, with which it is easily confounded, the pulse is usually rapid, being not rarely as high as 120–130.

Profuse continued sweating often occurs, causing sudamina. Herpes labialis is also seen. I have repeatedly seen unquestionable roseola on the abdomen and breast; Waller has seen the same.

Albuminuria is very common, and peptonuria is not rare.

The consciousness is often obscured at an early period; the patient pays no attention to things, or is furiously delirious.

As in acute septic endocarditis, so in general miliary tuberculosis, we can distinguish two chief types, the typhoid and the intermittent, corresponding to the fevers of those names. In the case of a man who had had a dry, fissured, red tongue, swollen belly, roseola, large splenic tumor, and diarrhœa, I found at the autopsy general miliary tuberculosis, old circumscribed foci in the lungs, splenic tumor, and old tuberculous ulcers of the intestine; and such cases are quite common in Zurich.

The diagnosis being sometimes very difficult, much value may be assigned to an observation of Weichselbaum, which has been confirmed by Meisel, Lustig, Ulacaris, and Doutrelepont, showing that tubercle bacilli circulate in the blood, and can be demonstrated in blood taken from living patients. The bacilli are few and scattered, and they require close search. Rütimeyer obtained splenic juice by puncturing a living patient with a Pravaz syringe, and in one case found tubercle bacilli; this organ is said by Weichselbaum and Lustig to be especially rich in the bacillus.

The local symptoms depend on the organ chiefly attacked. In many cases (especially when the lungs are affected), we have little else except severe coughing, continuing day and night, uncontrolled by narcotics, and soon followed by violent pains in the muscles of the breast and abdomen in consequent of the violent exertion.

There may be symptoms of bronchial catarrh—extensive sonorous and sibilant râles, rough, interrupted, or weakened vesicular respiration, and mucous expectoration. The sputum sometimes contains streaks of blood, or may be colored rusty brown, so as to resemble the sputa of fibrinous pneumonia. The disease began with spitting blood, in a case of Litten's.

Dyspnœa is often prominent; breathing is excessively accelerated, and often orthopnœic, while physical examination shows no cause for the acceleration. (Irritation of vagus fibres by miliary tubercle?) There are sometimes attacks of rapid breathing, having a superficial resemblance to asthma.

Cough and difficulty of breathing may be associated with great cyanosis, and this symptom becomes of especial importance when disease of the bronchi and alveolar spaces cannot be demonstrated.

There may be a total absence of local thoracic symptoms. The percussion sound is sometimes tympanitic, a sign that the tension of the lung-tissue is diminished, and in correspondence with this we may hear the cracked-pot sound, usually in the subclavian fossa.

The symptoms of old consumptive disease may be so prominent that we overlook general miliary tubercle of the lung. In this case, we find tubercle bacilli in the sputa, which would not be the fact in uncomplicated miliary tuberculosis, as bacilli are not expectorated until the nodules are softened and break into the air passages.

In miliary tubercle of the pleura, Jürgensen has heard peculiar crepitant sounds, distinguished from pleuritic friction murmurs by their soft character. In other cases, there is pleurisy of one side or both, and the general appearance of the disease is entirely that of exudative pleurisy; in this case the discovery of bacilli in the exudation would be of especial diagnostic value. The exudation is often hemorrhagic.

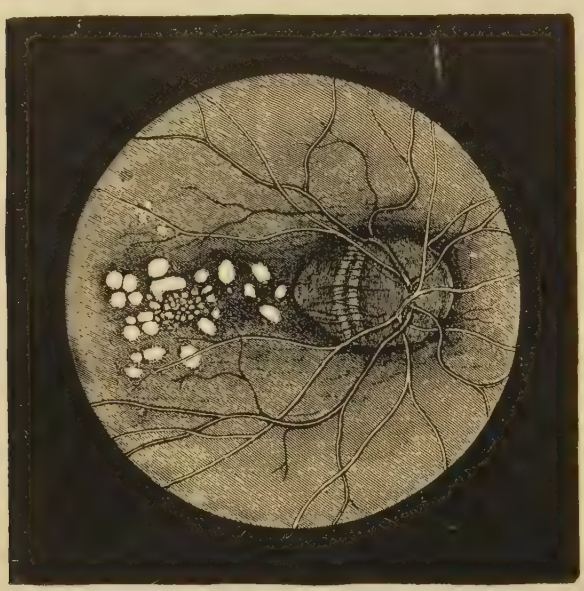
General miliary tuberculosis may be concealed behind the symptoms of pericarditis, peritonitis, or meningitis.

Traces of albumin in the urine do not necessarily point to miliary tubercle in the kidney, for this symptom is often found where there is only general infection and fever. Neither does the existence of tubercle bacilli in the urine allow us to assume miliary tubercle in the kidney; on the contrary, it would point to chronic tubercle with ulcerative destruction. Rosenstein refers many cases of anuria in children to miliary tubercle in the kidneys.

Precisely the same holds good in regard to tubercle bacilli in the stools.

Prominent importance must be attached, not only to the bacilli as

FIG. 59.



Tubercle of the choroid. After Jaeger.

demonstrated in the blood, but to those of the choroid, for the latter can easily be recognized during life with the ophthalmoscope as yellow washed-out spots (see Fig. 59). The symptom is not, unfortunately, a constant one, and can only be counted on when the disease extends to a great many organs.

Manz first saw choroid tubercle in the living subject; it was afterwards described by Graefe and Leber, Cohnheim, Fränkel, Steffen, Bouchut, and others. One should persevere in the examination from day to day, for it often happens that tubercle becomes visible in twelve hours. Sometimes we find only one, or a few tubercles; at other times they are very numerous. Cohnheim found fifty-two in one case at the autopsy. The tubercle must (as Graefe and Leber state) have reached a certain size before it is visible to the ophthalmoscope; it must also have caused atrophy of the superjacent pigment, and must lie close to the retina. Litten once observed the formation of a crater-shaped

depression, which at the autopsy corresponded to a central destruction of the choroid tubercle.

The course of general miliary tuberculosis is almost always unfavorable, and usually acute. Death does not commonly occur before the end of the second week. Wunderlich's case of death on the twelfth day, and Bressi's of death on the third day, are exceptions. From four to eight weeks is an average, though cases lasting several months occur.

Death may be preceded by symptoms of collapse, or excessive disturbance of breathing, or excessively high temperature, or meningitic symptoms, or symptoms of dissolution of the blood (uncontrollable epistaxis, bleeding from the gums), or by accidental complications, such as sudden rupture of the spleen (case by Aufrecht).

IV. Diagnosis.—We cannot usually be certain unless tubercle bacilli are demonstrated in the blood, or tubercles in the choroid. In the absence of both of these, we can only reach a certain degree of probability.

When caseous tubercle and ulcerative changes are visible in peripheral organs—lymphatic glands, tongue, pharynx, or larynx—we may have strong suspicions of general miliary tuberculosis.

The disease is most frequently confounded with:

a. Acute bronchial catarrh.

Pay attention to the severity of the general symptoms and the loss of strength, which is usually rapid.

b. Intermittent fever.

Notice the history. In the tubercular disease, the periodicity is apt to be less marked, and splenic tumor is often wanting, while in intermittent fever quinia produces rapid effects.

c. Typhoid fever.

In tubercular disease, the temperature is usually less elevated: distinct stages of the complaint are not seen; meteorism, roseola, and diarrhœa are usually absent; bronchitic symptoms at the beginning are more prominent.

d. Uræmia.

The mistake is easily made when the tubercular disease supervenes on nephritis. Rigal describes such a case.

e. Miliary carcinosis of the liver.

The differential diagnosis depends on the proof of a cancerous tumor of the liver, and the fact that the two diseases rarely exist together.

V. Prognosis.—This is unfavorable. Many think recovery possible, others deny it; it would certainly be a rarity. Periods of apparent improvement must not deceive us, for they are usually followed by a rapid return to the worse, and increased eruption of tubercle.

VI. Treatment.—This is purely symptomatic; we are often limited to antipyretics and narcotics.

8. *Tuberculous Inflammation of the Cerebral Membranes. Meningitis Tuberculosa.*

I. Etiology.—Tuberculous meningitis is simply a distinct variety of miliary tuberculosis. It may be a consequence of general miliary tuberculosis, or the miliary tubercle may be mainly confined to the soft cerebral membranes; the latter is the more unusual case.

There are two very distinct stages in the development of the disease. In the first, miliary tubercles appear upon the meninges without exciting inflammatory symptoms, while in the second the disease develops into tuberculous meningitis by the supervention of inflammatory processes. Morbid disturbance may be absent in the former case.

The causes are identical with those of general miliary tuberculosis; any tuberculous cheesy deposit may lead to general infection, and, in consequence, to an outbreak of general tuberculosis or to miliary tuberculosis of the soft meninges. A very frequent cause, especially in children, is the tuberculous cheesy change in lymphatic glands; most frequently those of the trachea and bronchi. Doutrelepont recently described the outbreak of tubercular meningitis after lupus; an observation of great importance, as presenting a kind of auto-inoculation, since the tubercle bacillus is found in lupus. There are a few cases in which no source of infection could be shown, so that it seems as if in some circumstances the meninges, like the fauces, larynx, intestine, and kidney, might be primarily affected. Compare etiology in the preceding chapter.

Children are much the most frequently affected, especially from the second to the sixth year; after that period, the disease grows more and more rare, and after the fortieth year it is exceptional.

The male sex is oftener affected than the female. The disease is more frequent in winter and spring than in warm weather.

II. Anatomical Changes.—As in purulent meningitis, the bones of the skull contain a great deal of blood, and the dura mater is very tense. Nodules of miliary tubercle may sometimes be seen on the surface of the latter, especially in the neighborhood of the middle meningeal artery, and also in the tissue of the dura itself. The sinuses usually contain much blood, in part coagulated. Raising the dura, we find its inner surface dry, and often dotted with little extravasations of blood.

The convex surface of the hemispheres is strikingly prominent, the convolutions are broad and flattened, the sulci obliterated.

The pia mater is usually very dry and deficient in lustre. Its blood-vessels are very full, and many can be traced to the finest ramifications. Very fine, transparent knots of tubercle may be seen here and there on the vessels; they are more distinctly seen when the pia is stripped off and held up against the light. Streaks of pus are seen on both sides of the larger vessels in the pia.

The changes in the pia and the subarachnoid tissue are usually well advanced at the base of the brain. The space between the optic chiasma, pedunculi cerebri, and pons, especially, is in a state of œdemato-purulent infiltration. In the course of the sylvian artery the tubercular eruption and the infiltration are usually very pronounced; this region should be carefully examined in cases that are not well marked. The inflammatory products usually have an œdemato-purulent character; less commonly they are sero-purulent or fibrino-purulent. The tubercles are not all gray and transparent; many have an opaque centre, and

others are undergoing cheesy metamorphosis. Diffuse cheesy tuberculous thickening and deposits in the pia are also sometimes seen.

The ventricles are often widely dilated, but not always with fluid, and not always in equal degrees. The fluid is usually serous, or serous and flocculent, seldom purulent. Cadaverous softening is frequent in the ependyma and neighboring cerebral substance, and single tubercles have been seen in the ependyma.

In the choroid plexus, purulent infiltration may accompany tubercles.

On the upper surface of the cerebellum, tubercles and gelatino-purulent infiltration of the pia are usually very marked.

Schultze notes the important fact that tuberculosis of the meninges, or tubercular spinal meningitis is usually found if that part is examined.

The substance of the brain and cord may be involved. Hemorrhage occurs; foci of inflammation and necrotic softening are found. The latter may be caused by tubercle compressing the blood-vessels and interrupting the circulation in a portion of the brain. A few tubercles also are found in the brain-substance.

In most cases, the disease is diffuse, though the base is usually more affected, and the convexity may be preferred. Local tuberculosis of the meninges is rarer. Such cases prefer the region of the arteria fossæ Sylvii; when the left artery is affected and the circulation is interrupted, they cause aphasia, alexia, agraphia, and hemiplegia of the right side. We would add that the left half of the brain seems especially predisposed to tubercular meningitis.

Fraentzel has reported a case of local tuberculosis in which the vessels of the choroid plexus alone were involved, the rest of the pia being unaffected. There was hydrocephalus. Death occurred within thirty hours, with attacks of loss of consciousness and twitchings of the face.

We do not enter on a microscopic description of tubercle in this place. Most authors state that it originates in the adventitial lymph-sheaths of the vessels of the pia, especially those of the veins, the endothelium being the part first involved. As it grows, it contracts the vessels proper. The internal and middle coats are often infiltrated in places with round cells. The tubercle may grow through the wall proper of the vessel; or in other spots it may contract the vessel and thus lead to thrombosis by compression. The true pathognomonic sign is always, not the giant-cell, about which there has been so much dispute, but the tubercle bacillus of Koch.

Tubercle has often been seen in the walls of the blood-vessel, especially in the inner coat. Ziegler states that the older view, of which Rindfleisch is the chief representative, of the genesis of tubercle from the endothelium of the adventitial lymph-sheaths, rests on a false interpretation, making the tubercle cells to be migrating blood-cells and proliferating connective-tissue cells.

A microscopic examination of the substance of the brain and cord shows that the tuberculous deposit often extends along the sheaths of pia mater into the brain and cord, producing diffuse infiltration, and often foci of softening in the vicinity.

The frequent preponderance of the basal affection has given rise to the term basal or basilar meningitis, as contradistinguished from the purulent meningitis of the convexity, but it is better to avoid these terms, as exceptions are too numerous to the supposed rule.

Tuberculous meningitis is also called acute hydrocephalus—a name which is unsuitable, because dropsy of the ventricles is sometimes absent in the disease in question, and is by no means exclusively confined to it.

III. Symptoms.—The clinical phenomena are essentially like those

of the purulent form, depending as they do upon inflammatory nisus in the blood-vessels, and increased intracranial pressure caused by the exudation. Symptoms of local lesion may be added, caused by necrotic or inflammatory softening of the substance of the brain. But purulent meningitis often advances as far in a few hours or days as the tuberculous form does in many weeks; the latter also may have remissions which resemble convalescence, but are almost invariably followed by exacerbations.

Premonitory symptoms are a marked feature, especially in children. They become cross, freakish, timid, and inclined to cry, sleep badly, dream a good deal, occasionally twitch, gnash the teeth or squint, lose appetite, and are costive.

I have often treated children that suffered from these rather undecided symptoms for several weeks before the first unquestionable meningitic symptoms appeared. I remember a case in which the only child of parents who married late in life was presented for my treatment; I was uncertain for weeks, until I saw yellow spots of tubercle in the fundus of the eye, and in a week the symptoms of stiff neck, high fever, and excessively rapid pulse had appeared and the child was dead.

Among the manifest symptoms, stiff neck, headache, giddiness, growing stupor, retraction of the abdomen (referred by Henoch to irritation of the splanchnic nerve and consequent contraction of the intestinal wall), and constipation are the chief. Add vomiting, which sometimes occurs only at the beginning, but in other cases is repeated many times a day during the entire sickness. Cases with meteorism and diarrhœa are rare. The hydrocephalic cry is rarer than in the purulent form. The pulse varies greatly in its rate, either spontaneously or after bodily emotion. The breathing is often very irregular in rhythm and depth. Sighing or sobbing breath often occurs. The Cheyne-Stokes phenomenon is more common than in purulent meningitis. The variations in temperature are numberless; there may be no fever at all—or fever only near the close—or only just before death—or feverish and typhoid conditions during the whole course of the disease. Temperature beneath the normal range has also been described repeatedly. Gnändinger reports several cases: in one, the rectal temperature fell to 28.6° C. before death. Rise of temperature after death also occurs.

As complications, we mention paralysis of the eye-muscles, pupil, face, and extremities. Twitchings are not uncommon. I have repeatedly seen paralysis disappear and return. Conjugate forced positions of the head or eye occur; also continued lateral decubitus with flexed hip and knee joints. Soporose patients not uncommonly make repeated motions of seizing or grasping with the same extremity. In three recent patients of mine, I found the patellar tendon reflex absent; and in two other observations, it was normal in the one and weakened in the other. The pulse is often retarded and unequal in force; afterward it usually becomes so fast that it can hardly be counted (irritation and paralysis of the vagus). The skin may display unusual irritability of the vaso-motors, so that slight irritation is enough to cause protracted redness. Roseola and herpes facialis also occur, and (in case of heavy sweating) sudamina. Icterus has been seen. The urine often contains albumin and peptones. Changes in the fundus oculi (choroid tubercle) and tubercle bacilli in the blood (Vol. IV. p. 212) are of great diagnostic importance. Neuritis and neuroretinitis occur simultaneously or independently; also apoplexy of the retina.

It is fashionable to divide this disease into stages—that of irritation, of increased cerebral pressure, and of paralysis. We think this incorrect and artificial; the symptoms of different stages almost always run side by side, and symptoms of paralysis and irritation frequently alternate in the same organ, as is seen in the pulse.

There are some singular cases, as when the disease opens with paralysis, or one or several attacks of aphasia, followed by paralysis of the face and extremities.

IV. Diagnosis.—The close resemblance between purulent and tubercular meningitis involves the danger of confusing them in diagnosis. Notice that the tuberculous disease develops more slowly, is more insidious in its progress, often has less fever or even none at all; add the evidence of hereditary tendency and previous scrofulosis, or tuberculous inflammations of the skin, bones, or joints. Some cases must remain doubtful, *e. g.*, tuberculous disease of the petrous bone is not necessarily followed by tubercular meningitis, for the purulent form may occur.

It is easy to confound the disease with typhoid fever, especially when, contrary to the rule, there is meteorism and diarrhœa, or roseola and enlarged spleen, for the rigidity of the neck may be found very fully developed in typhoid fever as a consequence of œdema of the pia mater. In this case, as in that of purulent meningitis, the diagnosis depends on the recognition of choroid tubercles, tubercle bacilli in the blood, and bacilli in the stools.

If the disease opens with paralysis or encephalitic symptoms, we may be inclined to infer embolism or thrombosis of the cerebral arteries rather than meningitis; here the important points are: rigidity of the nucha, and the state of the fundus oculi and the blood.

V. Prognosis.—The disease probably always ends in death. Recoveries are reported, but are doubtful. Dujardin-Beaumetz reports a recovery after choroid tubercle had been seen.

VI.—Treatment as in purulent meningitis. Holm recommends benzoate of sodium (1 part : 10 of water; tablespoonful every two hours) as a remedy (!).

9. *Tubercular Peritonitis.*

I. Etiology and Pathological Anatomy.—Of the causes of tuberculous inflammation, or of general tuberculosis of the peritoneum, we may repeat what was said under general miliary tuberculosis and tuberculous meningitis. The disease may develop as an independent disease, or may be the consequence of a widely diffused general miliary tuberculosis.

Miliary or submiliary gray nodules, rather than cheesy or partly calcified masses, are usually found. Inflammatory changes are often associated, many adhesions between the intestines and the abdominal viscera (constituting the so-called adhesive tuberculous peritonitis) and effusions, serous, or more commonly hemorrhagic, very rarely purulent. Extravasations occur in the peritoneum, so that the individual nodules are seen surrounded by a hemorrhagic area, or at a later stage (when the coloring matter of the blood has been altered), by black pigment. Bamberger mentions a case in which large clots of blood were found in the peritoneal cavity. Ascites is said to be common, but this has often been mistaken for serous peritonitis. If the tubercles are few, the examiner should look carefully at the omentum. In chronic cases, thickening and

crumpling of the omentum and mesentery occur, the omentum often forming a rolled-up cord which crosses the abdomen from right to left

Localized milary tuberculosis of the peritoneum must be distinguished from the general or disseminated form; it is found close to cheesy tubercles of the abdominal viscera, most frequently over tuberculous intestinal ulcers, in which case it often follows the lymphatics for long distances. The fact has no clinical importance.

II. Symptoms and Diagnosis.—Our diagnosis depends on our power, in the presence of peritoneal dropsy, to assign a tuberculous origin to such dropsy; and in doing this we chiefly lean on the facts of etiology, and on the discovery of tubercle bacilli in the blood and the peritoneal fluid. Vallin is wrong in supposing inflammation in the neighborhood of the navel to be characteristic of this disease.

The course is usually chronic, though at times it may become acute. There is pain, swelling of the belly, diarrhœa or constipation, loss of appetite, vomiting, and continued loss of strength. Fever may be present or absent, the contracted omentum may be felt as a tumor; even the adherent coils of intestine may give a similar impression.

There is especial danger of confounding this condition with cirrhosis of the liver and thrombosis of the portal vein, and errors cannot always be avoided in spite of all care, if the demonstration of the bacilli fails. The diagnosis of cirrhosis is favored by alcoholic excesses, enlargement of the spleen, alterations in size of the liver, and jaundice; that of peritoneal tuberculosis, by finding hemorrhagic fluid in exploratory tapping of the abdomen.

The curability of the disease is assumed by a few authors; I have just discharged a patient as comparatively cured, with a wrinkled and thickened omentum. The disease is usually chronic. Death usually comes as a consequence of progressive marasmus, or of asphyxia through increase of dropsy.

III. Prognosis. Treatment.—The prognosis is almost always unfavorable. The treatment is purely symptomatic. The above-mentioned patient from the Zurich clinic owed his cure to the long-continued use of sweat-cures and preparations of iron.

Appendix.—For tuberculous pericarditis and pleurisy see Vol. I., pp. 33 and 389.

10. *Scrophulosis.*

(*Scrofulosis.*)

I. Etiology.—The numerous clinical points of contact between this disease and tuberculosis have often been noticed, but Koch first proved that scrofulosis is but a special clinical form of the other, and owes its origin and development to the tubercle-bacillus. We are inclined to consider it as a chronic tuberculosis of the lymphatic glands, strongly predisposing the system to inflammations which are liable to become tuberculous.

Scrofulosis is especially a disease of childhood. It most frequently begins towards the end of the first dentition, or the end of the second year of life, and ends at the completion of puberty. Some of its consequences may persist through life.

This disease is rare in adults. It has been seen in prisoners living in gloomy,

narrow cells, forced to spend a long time in a sedentary way—the so-called prison scrofula. It is much commoner to find it beginning with the first dentition (about the ninth month), and sometimes earlier. Chaussier described a congenital form, in which a child was born with suppurating lymphatic glands (?).

The constitution is very important, as in the case of consumption. Faults may be inherited, congenital, or acquired. There are scrofulous, as there are consumptive families; many inherit both tendencies.

As regards congenital faults of constitution, we know that children are especially liable whose parents were old at the time of the birth of the former; or differed widely in age; or who were in a state of marasmus from consumption, cancer, tertiary syphilis, etc.; or were in a condition of wretched poverty; or were blood relations. Drunkenness of the father is said by many physicians to cause scrofula in the children. These facts are paralleled in the etiology of consumption.

Among the acquired faults of constitution which predispose to scrofula we would first name those which result from improper nourishment. This includes the case of children who have never had the breast, or good cow's milk, but have been brought up from the first on meal porridge and such indigestible foods. When children begin too suddenly to use adults' food, consuming abundance of potatoes, bread, and puddings with too little meat, scrofula easily occurs.

Bad diet and unwholesome surroundings greatly assist the development of the disease; hence it is frequent among the lower classes of laborers, whose children spend their first years in dark, dank, close cellars or attics, and seldom get into the fresh air.

Children of the better classes are not rarely attacked by scrofula when they are over-taxed with school work and mental tasks which interfere with the care of their health and with play in the open air.

There are several reports of endemic scrofula in institutes for the blind and deaf-mutes, but which diminished when the pupils were obliged to take daily walks in the fresh air.

Certain diseases of childhood, especially measles and whooping-cough, less frequently scarlatina and diphtheria, German measles and small-pox, have a bad reputation for causing scrofulosis. It has appeared subsequent to vaccination—a fact which has been quickly used to represent vaccination as the means of implanting diseases in well children. Of course, all these influences only convey a predisposition to scrofulosis; the infection with tubercle bacilli is needed to produce the disease, and in regard to the manner of communication in scrofula hardly anything is known.

We must further note that in cold, damp countries scrofulosis is especially common, and is frequent among persons (even adults) who remove from the tropics to temperate climates.

II. Symptoms.—Scrofulosis is to be specially feared for children who are predisposed by inheritance, or whose elder brothers or sisters have had it. It is also stated that children who have it often have had premature eruption of teeth (the normal date being being the ninth month).

Two forms were correctly distinguished by the older authorities—the torpid and the erethistic.

The torpid cases have a thick coat of fat and look spongy; the lips are thick and turned up, the lower part of the nose is thick and shapeless, or pear-shaped, and as it were pendulous. The patients are lazy,

have a stupid, clumsy, almost vulgar expression, and are usually not remarkably lively.

The erethistic cases have a delicate, soft, pale skin, with bluish veins winding over the forehead and breast. The hair is usually blond and soft, the eyes large, with bluish sclerotica, and possessing a peculiar swimming lustre. The teeth are long, bluish-white, and translucent. The mind is lively, sparkling, and quick to grasp. The face easily blushes with passing emotion.

The symptoms of scrofulosis vary greatly; surgery or ophthalmic or aural science may be more required than internal medicine. We shall refer only to such symptoms as belong to the latter domain; we cannot even enumerate the others.

Scrofulous (tuberculous) changes of the lymphatic glands are first betrayed by swelling, but not every swollen lymph gland is primarily tuberculous; for example, primary scrofulous inflammations of the skin may often be accompanied by secondary sympathetic non-tuberculous buboes.

Those most frequently affected specifically are the cervical and the submaxillary glands, which may become as large as a pigeon's egg, and by uniting may make bunches that protrude under the skin of the neck, producing deformity and causing a mechanical obstacle to movement of the head. As long as no further complications exist, the skin over them is natural and flexible. The glands are flattened, and not tender. They sometimes form a row of swellings along the sides of the neck, making a sort of chain.

Careful observation will often discover enlargement of the inguinal, occipital, and cubital lymphatic glands (the latter above the internal condyle of the ulna), which shows how erroneous it is to take the two latter as a sure sign of syphilis.

The internal lymphatic glands may become enlarged in precisely the same way, though this is rather unusual. The bronchial glands form examples. This is recognized by dulness over the manubrium sterni, sometimes even by slight swelling at that point, or symptoms of bronchial stenosis or paralysis of the recurrent nerve from pressure. Obstructions of the veins of the neck are sometimes noticeable (swelling of the veins and even slight œdema). Scrofulous (tuberculous) changes of the mesenteric glands are connected with symptoms of tabes mesenterica: the children usually suffer from uncomfortable diarrhœa with disgustingly offensive discharges, the belly is distended like a frog's, the mesenteric glands are often recognizable on deep pressure, the hunger is unappeasable, and yet the child emaciates day by day, becoming pale, hollow-eyed and hollow-cheeked, losing the hair, acquiring a withered, lean, and pendulous skin and a peculiar old look. Death often follows after progressive emaciation.

Scrofulous swelling of glands is usually slow in developing; but there are exceptions. I once treated a boy of six, in whom acute swelling of bronchial glands came on, and in five days produced severe disturbances; there had been no perceptible tumor previously.

The swellings of glands may recede spontaneously, or may soften and suppurate, usually beginning at the centre; the pus may break directly, or may form long fistulous passages. Redness and swelling of the covering skin, adhesion of the skin to the gland from periglandular inflammation, are external signs of this condition. Suppuration may be protracted, the fistulæ do not close, but form ulcers with undermined

swollen edges; and at last leave disfiguring cicatrices with radiating puckers, which may by their contraction cause deformity of the neck and impede its motion.

The tonsils are glands of this class which are often found in a state of chronic hyperplasia in scrofulous persons. This may cause a tendency to obstinate catarrh of the throat and follicular inflammation, disturbance of speech and breathing, and even asthmatic attacks.

Scrofulous disease of the skin most commonly takes the form of impetiginous eczema, affecting the face or the hairy scalp with especial frequency, and very often accompanied by inflammatory swelling of the nearest lymphatic glands—secondary or sympathetic bubo. There is little to distinguish this from eczema in non-scrofulous persons, unless it be its great obstinacy and great tendency to relapses. Eczema of the skin often attacks the neighboring mucous membranes, especially those of the nose and ear, where they become still more obstinate, and give rise to discharges and further inflammations. The converse also occurs: eczema of the skin caused by irritating discharges from the ear or nose.

Obstinate acne is often connected with scrofulosis. Lichen hardly ever occurs except in scrofulous persons; lupus is closely related to scrofula, as it is a form of tuberculosis of the skin. Lupus is a sort of late form of scrofulosis, usually developing after puberty. The tendency to chilblains is another instance of the great vulnerability of the skin of scrofulous persons.

Abscesses often form in the subcutaneous cellular tissue, which keep recurring, and may cause dangerous loss of strength. If they break outwards, ulcers that are hard to cure often form, sometimes crater-like, and resembling syphilitic ulcers of the skin. A part of these formations is tuberculous.

The mucous membrane of the nose is often affected, causing obstinate inflammation, frequently relapsing, and at last permanent. Many cases of obstinate cold in the head are connected with scrofulosis. Ulceration is not uncommon, and may extend to the bones of the nose. Impetiginous eczema also occurs, frequently forming the basis of relapsing erysipelas of the face. Obstinate angina and pharyngitis are also frequently due to scrofulosis. Bronchial and gastro-intestinal catarrhs are known to be frequent; a less commonly known complication is a seropurulent discharge from the vagina, which very obstinately resists treatment, and often causes inflammation and swelling of the labia, resulting in abscess or gangrene.

Scrofulous disease of the bones causes necrosis and caries, which may be due to tuberculous deposits. The vertebral column is very often attacked, causing kyphosis, gravity-abscess, perhaps peripachymeningitis, compression myelitis, etc. Or tuberculous inflammatory swellings appear on the bones of the fingers and toes (spina ventosa), or in other parts, as the ribs, sternum, or bones of the limbs. These are surgical diseases; so are the tuberculous affections of the joints which are known as fungus of the joint, tumor albus, or arthrocace.

The eyes are often affected in scrofula, though not in a specific way. There may be impetiginous eczema of the eyelids, ciliary blepharitis, catarrhal and phlyctenular conjunctivitis, keratitis, and often several of these in combination. These may cause trouble for the whole of life; as opacity of the cornea (leucoma), or adhesion between the cornea and iris (synechia anterior). Horner associates lamellar cataract with scrofula.

The external meatus of the ear is sometimes inflamed and discharges; sometimes the middle ear is inflamed in connection with throat-catarrh; and sometimes the petrous bone is tuberculous, which destroys the organ of hearing permanently or leads to thrombosis of the sinuses or inflammation of the meninges and the brain.

Scrofula is a chronic disease, often lasting many years. The changes often begin in one organ, and extend to others successively. In protracted cases, leucocytosis has been observed; Horand states that he has seen the red corpuscles of the blood lessened in size. Remissions and exacerbations are common; the patient is often worse in winter when shut up in the house, and better in summer.

Among the complications, miliary tuberculosis is the most dreaded; this, or tuberculous meningitis, may suddenly appear after apparent recovery, and at the autopsy the bronchial glands are apt to be in the cheesy-tuberculous state.

In case of protracted suppuration, amyloid degeneration of the great glands of the abdomen may occur, known by the hard swelling of the liver and spleen, albuminuria, and œdema. Fatty liver also occurs.

III. Anatomical Changes.—These are composed of various elements, some specific, others not. We do not here describe the latter. The specific inflammation of the lymphatic glands is essentially based on the development of the tuberculous foci containing the bacillus, often in the interior of giant cells; in cheesy foci, the bacillus is usually wanting. The bacillus is usually in small numbers, and this may aid in giving a local character to the affection. The recently attacked glands are gray and hyperæmic, but in older ones the cheesy process becomes prominent.

IV. Diagnosis.—This is usually easy if we regard the totality of the symptoms rather than any single one, or if we discover the bacilli.

V. Prognosis.—This is comparatively good. Most patients escape with life, but we must remember that disfiguring scars, deformities of the bones and joints, severe spinal diseases, or irreparable injury of special senses may occur, and that general miliary tuberculosis is a threatening possibility.

VI. Treatment.—The etiology shows us what to do for prophylaxis. Blood-relations should not be encouraged to marry; marastic or consumptive or tertiarily syphilitic patients should not get children. The physician should forbid women of scrofulous or consumptive families to nurse their own children, especially when they themselves show signs of these diseases, or when they are younger than eighteen, or are anæmic. Let wet-nurses or good cow's milk be then substituted. The food of children must be strictly cared for, and plenty of exercise in the open air allowed.

After the disease has appeared, general and local treatment will usually be required.

The general treatment concerns the nutrition and occupation of the patient, the diet, and exercise in the open air. In summer, let the patient live in the mountains, or better, by the sea; in winter, in Meran, Nice, or other mild climates. Mental overwork should be forbidden.

Cod-liver oil is a very important remedy. Give about a dessert-spoonful, half an hour after the first breakfast, and the same amount after supper; to older children, from this amount to twice as much. A peppermint lozenge may be taken directly afterwards. The remedy needs to be taken for months or years; it should be suspended for a week or two,

every four or six weeks, to prevent unconquerable disgust. In the hot summer months, it will be best to leave it off, as it is liable to injure the appetite. The ferrated oil is desirable for pale children; the iodated oil seems to us to have no special value.

It is not known what gives its peculiar value to cod-liver oil. Many have incorrectly ascribed it to the very small amount of iodine contained. The secret is probably in the fact that the oil is very easily absorbed and digested. It is commonly supposed to be useful only in the erethistic form, but we do not accept this unconditionally.

The preparations of iodine and iron, and iodine and iron baths, are next on the list.

For the frequent combination of scrofulosis with rachitis, we prefer the powder given at p. 94, Vol. IV. Otherwise we advise

℞ Syr. ferri iodati.....fl. ʒ ij.
Syr. simp.....fl. ʒ ss.
M. S. One teaspoonful three times a day after eating.

Or,

℞ Ferri iodati saccharati.....gr. ½
Sacchari albi.....gr. viij.
M. f. pil. no. x.
S. One pill, three times a day.

I have lately made trial of Fowler's solution, and am well pleased; I give it mixed with equal parts of bitter almond-water, three to five drops of mixture three times a day, after meals.

For brine baths, dissolve two to five pounds of sea-salt or common salt in a full bath at 28° R., and let the patient remain in it twenty or thirty minutes daily; after which he should rest an hour in bed or on a sofa. In poor families the bath can be used several times by adding boiling water and some more salt when used.

Natural brine baths are preferable, and the sea-bath is the best. Seaside hospitals have long been in use in Italy, France, and England, for poor children, with excellent results. Bergeron states that granular swellings, cold abscesses, scrofulous ulcers, and joint-diseases are much improved by a residence at the sea, while bone-tuberculosis is not influenced, and eczema and blepharitis are made worse.

There is no lack of brine-baths ("soolbäder"). We mention the best known: Arnstadt (Thüringen), Bex (Switzerland), Cannstadt (Wurtemberg), Dürkheim (Pfalz), Frankenhausen (Schwarzburg-Rudolstadt), Gandersheim (Brunswick), Gmunden (Austria), Hall (Tyrol), Hamburg (Prussia), Juliushall (Brunswick), Ischl (Salzkammergut), Kensington (Bavaria), Königsdorff-Jastrczemb (Silesia), Kösen (Thüringen), Köstritz (Gera), Kreuznach and Münster a. Stein (Rhenish Province), Nauheim (Hesse-Darmstadt), Neuhaus (Bavaria), Rheme-Oeynhausen (Westphalia), Reichenhall (Bavaria), Rheinfelden (Aargau), Rothenfelde (Hanover), Salzdetfurth (Hanover), Salzhemmendorf (Hanover), Salzschlirff (Hesse), Salzufeln (Lippe), Salzungen (Thüringen), Schweizerhalle near Basle, Soden a. Taunus (Prussia), Soden a. d. Werra (Hesse-Cassel), Sodenthal (Rhenish Bavaria), Sulza (Thüringen), Salzbad (Alsace), Sulzbrunn (Bavaria), Weisbaden (Nassau), Wittekind (Prussian Saxony).

Among the iodine-springs we name Tölz and Adelheidsquelle, both in Bavaria.

Preparations of baryta, antimony, and mercury are among the obsolete remedies. Decoction of walnut leaves is recommended by recent authors.

Local treatment is not given in this place; if suitable to the character of this work (*e. g.*, as in eczema) it is given elsewhere. For glandular swellings Novello has recently recommended tincture of tapya (one to ten drops three times a day). I have seen good results from chloride of gold and sodium (gr. $\frac{1}{12}$–$\frac{1}{6}$, three times a day in pill). Kapesser advises the rubbing with soft-soap (schmierseife), which is also praised by others (Hausmann); from one-half to one and one-half tablespoonfuls are dissolved in lukewarm water, and the back and limbs are wet-rubbed with it twice a week for ten minutes, and then washed off with water.

For tabes mesenterica we advise careful diet, cod-liver oil, decoction of quinia, and styptics, especially colombo and cascarilla.

PART II.

SYPHILIS.

(*Lues Venerea.*)

Public attention was first called to this disease at the end of the year 1494, when it spread as an epidemic among the troops of Charles VIII., who were then besieging Naples. The pest very soon travelled thence to Spain, France, Germany, and other lands. Many have believed that the disease was previously unknown, and took its origin from floods, from an unfavorable conjunction of the stars, from protracted rains, and from the practices of the abandoned soldiery with their glandered horses. Many said it was brought from America by the sailors of Columbus; others that it was an offshoot of leprosy. None of these views have prevailed; it is now with reason believed that syphilis was known in the oldest times, but became common among Charles VIII.'s military hordes whence it spread abroad.

It soon became known that sexual intercourse was the means of transmission in almost all cases. When the clergy, and even secluded nuns, became affected, it did not seem necessary to suppose that the disease might exceptionally be carried through the air.

The disease had at first a great variety of names; it furnished different nations with the means of giving each other a satirical thrust, as the names French, Neapolitan, Spanish, German disease, etc., show. The name of syphilis originated with Fracastorius in 1521, who invented and verified the legend that a herdsman of King Alcithous named Syphilus was punished by Apollo with the disease for refusing to pay him due honors.

Gonorrhœa, soft chancre, and syphilis were at first considered one and the same disease; it was thought that the poison might cause purely local injury to one person, while another's constitution was completely undermined. This erroneous doctrine lasted till near the middle of our century. A correct view is obviously of far more than theoretical importance in this case.

Balfour in 1767 first attempted to give to gonorrhœa a separate position as a local disease, but his views were not adopted. John Hunter in 1786 inoculated a well man with the purulent secretion of gonorrhœa

and produced syphilitic disease; of course, the case must have been one of masked chancre in the urethra. Although Benjamin Bell (1793) took Balfour's side, it was reserved for Ricord (1831) to give to gonorrhœa its true position.

Ricord's scheme, however, identified the soft chancre and syphilis. Two of his pupils, Bassereau and Clerc, separated them, giving to soft chancre a place with gonorrhœa as a local disease. These views are not yet universally adopted. The "unicists" still believe that general symptoms sometimes follow soft chancre.

Rollet explained a part of the difficulty by the "mixed chancre"—a soft ulcer, which in a subsequent coitus is infected with syphilitic virus. It seems to heal within a short time, but after some weeks symptoms of syphilis follow.

The symptoms of syphilis have been divided into those of the primary, secondary, and tertiary periods (Ricord). The exceptions to this arrangement are frequent, as is the case with other diseases that have a typical course; but the scheme is of great use in practice. While the primary period is essentially confined to the development of a hard chancre, the secondary includes a great variety, mostly of superficial affections, on the skin and mucous membrane; while the tertiary is signalized by gummata, which also develop in the interior of viscera—whence the term visceral syphilis.

There is also an hereditary form of syphilis.

1. *Acquired Syphilis in the First and Second Stages.*

I. Etiology.—Syphilis is a highly contagious disease. It cannot be doubted that the poison exists in the blood of the patient, for when such blood is placed under the skin of well persons, intentionally or otherwise, they become syphilitic, almost invariably. One source of contagion, therefore, may be the passage of blood directly from abrasions of the genitals of one party to similar abrasions in the other, during coitus.

The secretions of all cutaneous syphilitic alterations of the first and second stages are contagious. Contagion by products of the third or gummous stage does not seem to occur, though views are not united on this point. During coitus, the secretion of condylomata lata may penetrate into wounds of the well person; and in kissing, a similar disease of the lips may infect a crack in the lips of the well person.

The physiological secretions of syphilitic persons, as tears, nasal or bronchial mucus, sweat, saliva, milk, and urine, are not infectious of themselves, but may accidentally acquire infectious properties. If, for example, the secretion of broad condylomata of the fauces mingles with the saliva, the mixture is not free from danger.

Two physiological products are exceptions to this statement—the semen and the ovum. Both are almost always infected in syphilitic persons; it is matter of experience that such persons have no healthy children while they are under the influence of the disease.

Sexual intercourse is the most frequent, though not the only external means of contagion. In many cases it occurs by accident.

Among these accidents are kisses; the use of vessels for drinking and eating, of pipes and cigars previously employed by syphilitic persons, may also communicate the disease. Surgical instruments used unawares upon syphilitic patients and afterwards, without disinfection, upon well persons, may communicate it. It has repeatedly occurred in catheterism of

the Eustachian tube, and also after the use of the lancet or the cupping knives. The use of razors previously employed by syphilitics has repeatedly communicated the disease. In Jewish children it has occurred after circumcision, if the operator was syphilitic, and after operating applied his lips (fringed with syphilitic growths) to the wound to check the bleeding. Bites and scratches from syphilitic persons have transmitted the disease. In glass factories, a sort of epidemic has repeatedly occurred, owing to the passage of the blow-pipe from mouth to mouth. This does not nearly exhaust the list of accidents.

Physicians and nurses are liable to accidental infection. There are many sad cases in which physicians have examined patients, whose disease they may not have been aware of, and have been infected through scratches or cuts on their fingers. Such cases are often not understood for a long time.

Midwives also are often infected while conducting cases of labor, and themselves become sources of wide-spread infection.

Bardinet reported that in 1874, in the town of Brive, a midwife became infected in this way, and in eight months communicated the disease not only to her husband, but to all the women (more than one hundred) whom she attended in labor. Jean Beyer described a similar epidemic in 1725; forty women were infected in four months, and communicated disease to their husbands and children—in all eighty persons. Bleynic gives a like case from the arrondissement Rochechouart about 1850–60; the origin of the trouble was a syphilitic midwife's practice of touching the navel of the new-born child with her saliva.

Vaccine syphilis is deserving of special mention. The opponents of compulsory vaccination speak of the danger of infecting children with the lymph taken from a syphilitic child. The pure contents of such a vesicle, however, do not infect; they do not acquire infectious properties until enough blood is mingled to be visible with the naked eye. It is against well-known rules to vaccinate with lymph which has a tinge of bloody color. A number of physicians who have been charged with communicating syphilis in this way have, in self-defence, asserted that they had observed the rules; this has led to a doubt whether the pure lymph is always free from danger. Microscopic examination of the contents of vaccine vesicles always shows single blood-corpuscles, but that does no harm as long as blood cannot be detected by the naked eye.

No prudent physician will use a child to vaccinate from, if there is the slightest suspicion of hereditary syphilis, even though the danger of spreading be very slight. This danger can be much diminished by forbidding the use of healthy children under six months old as vaccinifers, for the first symptoms of syphilis may appear later than the third, but hardly later than the sixth month.

With the exception of the hereditary form, syphilis mostly affects adults. If children have it, the causes are inheritance, or accidental infection, or violation, or exceptionally, carelessness in vaccination. Men have it oftener than women, not only because society allows men greater laxity of morals, but because one infected woman may communicate it to many men. Climate and geographical position have no influence; the poison flourishes wherever it is carried and has access to the vessels conveying the fluids of the body.

The nature of the poison is unknown, but of late years more and more believe it to be due to schizomycetes. Klebs observed in the juice of the tissue of a hard

chancre, besides round cells, certain rods, 2 to 5 μ (1 μ=0.001 mm.) long, moving slowly, which he cultivated with success and transplanted to apes. Baumann, and Martineau and Hamonie have described something similar, while Pisarewski and Aufrecht report round schizomycetes, micrococci. By a complicated process, Lustgarten has lately shown the regular presence of syphilis bacilli in syphilitic sclerosis, condyloma, gumma, and syphilitic excreta; experiments in culture and inoculation are wanting. His statements are confirmed by Doutrelepont and Schütz and De Giacomi, who give simpler methods. Doutrelepont states that he has lately found them in the blood and preputial sebum of patients. Alvarez, Cornil, and Tarvel say they have found them in the smegma of well men also. Lustgarten found his syphilis bacilli always inclosed in round cells, never free. The subject is not yet in a condition for decision.

Böck and Scheel found syphilitic material active when diluted with one hundred parts of water; with five hundred parts it had lost its activity. When preserved in lymph-tubes it loses its power in a week. Cold does not change it, but a temperature of 40° R. destroys it.

It seems uncertain whether syphilis occurs in animals. It is said to have been observed in hares, and to have been produced artificially in apes. Some claim to have produced it in other animals by inoculation (?).

After one attack, a person is protected against another, but cases are known in which reinfection occurred after some years.

Syphilis and other infectious diseases (typhoid, pneumonia, erysipelas, etc.) do not exclude one another. It is often noticed that the symptoms of syphilis recede, while those of other infectious diseases are prominent, and in a few cases they do not reappear. A relapse of syphilis during erysipelas has been known to spare the parts where the latter disease existed.

Syphilis is endemic in many regions, as the coast of Jutland, Holstein, Pomerania, and also in the interior. This state is promoted by crowded quarters, lax morals, and indifference to disease. In large cities, especially seaports, it is especially common, as sensuality and extravagance are rifest there.

II. Symptoms.—Syphilis is essentially chronic. Cases that have a short course, and sometimes cause death soon, are exceptional. Relapses are common, sometimes returning at intervals during life.

The following is an outline of the course of the disease. Directly after infection, nothing is perceived for some weeks (the so-called stage of incubation); after which a hard knot or ulcer with cartilaginous edges appears at the point of infection—the ulcus durum. The neighboring lymph glands swell at the same time. Then some weeks more pass without further changes; many call this a second stage of incubation, but it is only the time required for the spreading of the poison in the general circulation. A series of exanthemata now appear, usually called syphilides, and forming Ricord's secondary stage; the first stage being that of the development of the hard chancre.

A tertiary period often occurs, especially in neglected cases. While the broad condyloma is the chief and the commonest affection of the skin and mucous membrane in the secondary stage (called the condylomatous stage), the gummous tumor of the skin and mucous membrane, often found in internal viscera, belongs to the tertiary, and by its breaking-down causes dangerous symptoms.

A stage of marasmus and sequelæ sometimes follows.

The stage of incubation is on the average from three to four weeks; a longer time is known; a shorter is less common. The duration is most accurately known in cases where syphilis is intentionally produced by in-

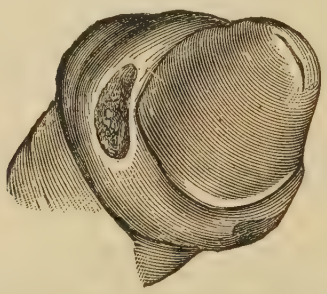
oculation; the shortest time was ten days, the longest forty-four, but Sigmund has observed as long as fifty-six days.

The hard chancre (ulcus durum), also called primary or initial sclerosis, or Hunterian chancre, is the first manifest alteration. It is most commonly seated on the sexual parts; in men on the outer or inner surface of the prepuce, the anterior border of the prepuce, the skin of the penis, the point of junction between the inner lamella of the prepuce and the coronary sulcus; less frequently on the frænum, meatus, or in the urethra. In the latter case it is called masked or urethral chancre. In women, it is most frequent on the labia majora or posterior commissure, rarely on the prepuce of the clitoris, the mons, or vaginal part of the uterus.

As the hard chancre merely shows where the poison first entered and began to act, there is no difficulty in understanding how it is found on such spots as the lips, etc. American physicians (Taylor, Knight) have lately spoken of chancre of the tonsils as not very rare.

In typical cases, there is a single hard lump, usually of longish form, resembling cartilage in consistency, and quite sharply defined. Its size

FIG. 60.



Ring-shaped hard chancre of the prepuce, with superficial ulceration. Author's observation. Zurich clinic.

may exceed that of a bean. In hard ulcers on the inner lamella of the prepuce, we can usually feel the hard lump plainly by pressing between two fingers. The skin over the lump grows thinner and redder, and gets a peculiar glazed look. A slight secretion is sometimes found on the surface of the lump, which is serous and dries to a thin scab (see Fig. 60). Under pressure, the lump is not sensitive or very slightly so; no bleeding occurs, as in soft chancre.

Sometimes coarser processes occur; deep and even crater-shaped loss of substance, with the edge retaining its cartilaginous hardness; or gangrene or phagedæna, which may cause the loss of considerable portions of tissue.

The fact that hard chancre is almost always single is very important as distinguishing it from soft chancre.

Deviations from the form described occur. If rhagades change to hard chancres, the shape is longish and flattish. On the glans we often see flattened chancres. They seem of the hardness of parchment, thin like paper, and may easily be overlooked (ulcus durum foliaceum, s. papyraceum, s. pergamentarium). Hard chancre often develops in a hair follicle, often taking a papulous form, easily confounded with acne by the inexperienced.

Hard chancre, left to itself, may last many months unchanged.

When it begins to disappear, the centre becomes more and more depressed, like a pit or a navel. Absorption often is complete and leaves nothing behind, but white cicatrices surrounded by a brownish ring of pigment may arise if ulceration was present.

Absorption is often imperfect, leaving a red thickened place, less hard than before, which may at times swell and harden again—a change which may precede other relapses on the skin and mucous membrane. The hard ulcer may have relapses like other symptoms of syphilis—ulcus durum redux.

Among the complications we name phimosis, balanitis, posthitis, balano-posthitis, and paraphimosis, which easily occur when the hard ulcer is seated on the anterior or posterior point of attachment of the inner lamella, in the coronary sulcus, or on the glans. In phimosis and paraphimosis, these complications often have to be relieved by cold applications and injections into the preputial sac, before the ulcer is seen. When a hard ulcer is seated at the point of junction between glans and prepuce, internally, we may, by drawing back the prepuce, easily see how paraphimosis originates: the ulcer often snaps back like a valve, as the cartilage of an upper eyelid will do when we evert it. Hard ulcers on the meatus cause tickling and pain, and sometimes mechanical interference with urination. Ulcers in the urethra cause a purulent discharge which may be mistaken for clap, but pressure on the urethra commonly detects a hard knot. In women, the chancre often loses its specific character in a short time, changing to a broad condyloma, so that we then have general syphilis, and the primary symptoms seem wanting.

The histology of the initial sclerosis is explained by Biesiadecki, Auspitz, and Unna. The blood-vessels of the cutis are to a certain extent the point of origin. An accumulation of round cells first forms in the adventitia. Some of them pass into the surrounding connective tissue and infiltrate it. In the mean time thickening and sclerosis of the connective-tissue fibrils, and proliferation of the cells, take place. By degrees, thickening and nuclear proliferation attack the middle and inner coats of the arteries. The endothelium swells, projects into the vascular cavity and contracts it, and acute endarteritis obliterans results. The lymphatics are attacked later, showing multiplication of nuclei in their adventitial tissue, but retaining their wide-open passage. These changes begin in the upper vascular regions and gradually extend deeper.

The epidermis is not untouched. The papillæ are first observed to extend deeper into the skin than is normal. The cutis-tissue becomes increasingly infiltrated with round cells, and the individual papillæ are laterally compressed and attenuated. Single round cells pass into the deeper layers of the epidermis, and at last nests of round cells form. The syphilis bacilli may form an especially important component (see Fig. 61).

While the hard chancre is developing, very remarkable changes also occur in the nearest lymphatic glands, and often in the vessels. In chancre of the penis, the lymphatics of the dorsum penis are often changed into hard, round, often knobby cords, not especially sensitive to pressure between the fingers. In very unfavorable cases, they may suppurate.

The inguinal glands swell; not one or a few, but a considerable number of them. One side is often more affected than the other, or one gland much more than another. Often, only that side is affected which corresponds with the location of the chancre. Single glands often grow to the size of a walnut and larger, forming in combination large lumpy bundles which can be felt through the skin. They are not sensitive to pressure; the skin above them is neither hot nor red. This is the

multiple indolent bubo; if patients walk about much, or poke and rub the inguinal region, secondary inflammation may supervene.

Indolent buboes often last for months and years; their remains often persist for a lifetime; a careful examination will show that remissions and increase in the swelling are common. Other changes are the caseous, calcareous, and amyloid degeneration (Virchow).

If hard chancre is situated on other parts than the genitals, the nearest lymphatics also swell. In the case of the lips, we find a painless lymph gland, greatly

FIG. 61.

Syphilis bacilli in an initial sclerosis. After Lustgarten.

swollen, either behind the angle of one jaw, or (as in three cases of mine) under the chin, forming a projection like a pigeon's egg. In the case of the fingers, the corresponding cubital or axillary glands swell, etc. These points must be applied in doubtful cases for the diagnosis of hard chancre.

After the formation of the hard ulcer and adjacent indolent buboes, a period of repose ("second period of incubation") seems to follow, not at all fixed in length, but averaging six or seven weeks; so that new symptoms may be looked for from the ninth to the eleventh week after impure coitus. In a few fortunate cases, the disease seems to end with

the first symptoms. Other patients have undergone some change during the second period of incubation; they are pale and depressed, often seem hypochondriacal, and, in a general indefinite way, feel sick.

The secondary period often begins with an eruptive fever, which may even be preceded by a chill, or several slight chilly sensations. The fever may last longer than a week, is usually remittent, and sometimes is almost typhoid. Enlarged spleen and red spots (roseola syphilitica) are not uncommon, and may lead to the diagnosis of typhoid. Abundant roseola, with erythema, may be mistaken for scarlatina or measles. Albuminuria occurs in twelve per cent of the cases (Fürbringer); tube casts, round cells, and red blood-corpuscles are found in the urine. Mercurials generally put the symptoms quickly to flight. Acute cases are known, of the kind called "syphilis maligna acutissima" by Guibot; the symptoms come in hasty succession, are very marked, and involve great danger to life. Weakly constitutions and external misery favor the development of this form. Bälz has reported hemorrhagic syphilis, with bleeding on the skin, from the nose, air-passages, stomach, intestine, and kidney. One patient died with such symptoms in ten days.

In the secondary period, indolent swellings of the lymphatic glands become more general, often including almost all the peripheral glands. In other cases, the swelling is confined to definite parts of the body, depending on chance—but parts exposed to pressure or irritation by the clothing, or in the neighborhood of accidental wounds, are more liable to be affected. In scrofulous patients, the swelling may be enormous—the so-called strumous bubo. Internal lymphatic glands seem to be also subject to swelling.

It was once thought that swelling of lymphatic glands of certain regions occurred only in syphilis, and was, therefore, of diagnostic value; this includes the enlargement of occipital and cubital glands; but the point is not sustained.

The histological changes of indolent buboes consist of thickening of the trabecular connective tissue and increase of the cellular elements. In the lymph sinuses the endothelium is swollen, and its nuclei are increased. The adventitia and media of the vessels are infiltrated with round cells. The capsule of the lymph glands is usually thickened.

Absorption (spontaneous or under treatment) implies fatty change of the cells and gradual absorption of the detritus. The cheesy and chalky changes often exist, and even suppuration may occur sooner or later.

The secondary stage is further marked by alterations of the skin and mucous membranes, to the former of which the term syphilides is (unjustly) restricted, although the changes are fundamentally the same in both.

The cutaneous disorders include erythematous, papular, vesicular, and pustular exanthemata.

Of the erythemata (which usually form the first eruption) roseola is the commonest. It is composed of red-brown spots, from the size of lentils to that of the joint of a finger, and most numerous on the skin of the trunk. They are found also on the extremities; on the face, scarcely except at the edge of the hair. At first they become white when pressed with the glass pleximeter; but as they grow older, the color seen on pressure is yellowish or yellowish-brown, showing that there is exudation in the cutis and not merely hyperæmia. The number of spots varies greatly. The same is true of the duration; under specific treatment

they sometimes disappear in a few days. Yet I have often seen the roseola become more distinct in the first days of treatment by inunction, disappearing very quickly afterwards. Slight desquamation may follow their disappearance.

In examining for roseola, let the patient strip and stand for a minute or two; the effect of the air is to contract the blood-vessels of the sound parts of the skin, and bring the eruption into relief. Do not confound the common venous mottlings with syphilitic roseola.

Diffuse syphilitic erythema is much rarer than the circumscribed roseola, and usually lasts but a few days.

Condyloma latum is the chief of the papular syphilides, and the most important for diagnosis. It is so regularly met with that Ricord gave its name to the secondary stage of syphilis.

In men, it is most frequent on the penis and scrotum, more rarely on the glans and inner lamella. In women, it is especially frequent on the labia majora. In both sexes, it is frequent on the arms, the inner surface of the thighs, the inguinal flexure, the navel, the folds of the breast, axilla, angle of the mouth, naso-labial groove, eyelids, and even the ears. They also appear in the bed of the nails, and between fingers and toes (ulcers between fingers and toes may always be suspected as syphilitic).

Where skin lies against skin, we often find broad condyloma duplicated on the two opposing surfaces. We sometimes can show that such a sore began on one surface and thence infected the other. The same is the case, especially, on the labia majora, the inner surface of the thigh and labia or scrotum, the anus and the mammary folds. The secretion is highly contagious; when inoculated upon a healthy person it causes a hard chancre followed by general syphilis, but on a syphilitic person it causes pustules and afterwards ulcers that are unmistakably like soft chancre.

Broad condyloma, fully developed, presents a flat elevation of the skin, covered with a greasy, gray coat, often having a disagreeable rancid smell. It sometimes covers large surfaces. It frequently causes itching and burning of the scrotum, pain in the anus on defecation, with itching and heat, etc.

Broad condyloma often transmits the disease accidentally. On the lips, it may communicate it by a kiss, or the use of drinking or eating vessels, or pipes. On the nipple, it may infect a nursling.

This eruption does not appear as such at once, but originates by degrees from broad papular elevations of the skin, of a reddish or brownish color, originally covered with moist epidermis. The change to condyloma is due to the influence of the position in which they are found, which keeps them warm and moist, and macerates and throws off the epidermis. When a cure occurs, the greasy secretion disappears, the surface becomes smooth, and brown-red elevations appear, which gradually disappear, often leaving brownish or bluish discolorations, colored by remnants of pigment from extravasated blood, and not parting with the color when pressed.

The histology of broad condyloma includes infiltration of the cutis with round cells, dilatation of the blood-vessels, penetration of the adventitia with round cells, and proliferation of the papillæ of the cutis; also the epithelial papillæ of the rete Malpighii press farther forward, and the epidermis disappears. Aufrecht and Lustgarten claim to have found micrococci. Aufrecht describes

cocci which generally lie two together (diplococci), rarely in threes, and are colored intensely by fuchsine; Lustgarten showed bacilli.

Syphilitic lichen and psoriasis are also included in the papular eruptions.

Syphilitic lichen presents groups of small, brown-red knots, of the size of lentils; syphilitic psoriasis, large flat knots, covered with thin scales of epidermis. Thick scales with the lustre of mother-of-pearl or asbestos, as in psoriasis vulgaris, are rarely seen. The syphilitic form of psoriasis (contrary to the non-syphilitic) usually avoids the favorite spots of the latter, extensor side of elbow and knee, and prefers the palmar and plantar surfaces and the volar aspect of the fingers. Some even say that psoriasis in the palm and sole is always syphilitic.

Psoriasis on the palms and soles often departs from the usual type. We first see red spots shining through the epidermis; these continue to rise, the epidermis grows thin, and at last falls out in the region of the papule almost as if cut with a punch, while the latter shows a surface at first shining, afterwards scaly. In other cases, psoriasis has the form of extensive horny thickening of the epidermis, which has been called psoriasis cornea.

Vesicular syphilides are not very frequent in acquired syphilis; they are known as varicella syphilitica and pemphigus syphiliticus. The former presents vesicles, more or less numerous and scattered, of the average size of a lentil or pea, and usually surrounded by a reddened areola; in the latter, the vesicles are larger, and often contain a clearer and more serous fluid. They are found rather often between the fingers and on the palms and soles.

The pustular syphilides include acne and impetigo or ecthyma. In syphilitic acne, there is inflammation of the sebaceous follicles of the skin; in impetigo and ecthyma (pedantically separated from each other) there are large pustules, which leave cicatrices when they heal. The latter afterwards become white, and leave a mark on the forehead which during the remainder of life reveals to the experienced eye the previous occurrence of syphilis.

The syphilitic affections of the mucous membrane closely resemble those of the skin. The earliest and most constant is angina of the fauces. We usually find the fauces of a dark-red or livid tint, with swelling and increased secretion, which is often bounded very exactly by the line of the hard palate. The latter point has been suggested as a diagnostic mark of syphilis. I have often seen well-developed roseola on the mucous membrane of the fauces and mouth, consisting of red spots and circumscribed hyperæmia, like that of the outer skin. The broad condyloma is important; it is most commonly found on the tonsils, but occurs also on the soft palate, tongue, and inside of the cheek and lips. In smokers, it is often found on the lips and tongue. People with bad teeth often have it on the tongue, inside of the cheek, etc. It consists of elevations of the mucous membrane, glistening like mother-of-pearl, grayish or bluish white—termed plaques opalines. Others call them mucous patches. Continued destruction of the superficial layers may lead to loss of substance and bleeding; the former may cause deep sinuous ulcers of the tonsils, with subsequent cicatrization, and may even cut off the uvula.

The above affections do not by any means complete the list. There are other cutaneous diseases. In many cases the skin loses its usual

brilliancy and fulness and becomes brittle, with a tendency to crack and scale off.

The hair often drops off, so that in a short time, not only the head, but the parts around the eyes, the chin, lips, and pubis, become nearly bald—defluvium capillitii sive alopecia syphilitica. It is a popular notion that early baldness is a sign of sexual excesses. Specific alterations also occur in the nails; sometimes inflammation of the fold and bed of the nail (paronychia s. paronyxis syphilitica), sometimes an affection of the substance of the nail (onychia s. onyxis syph.). The former are broad condylomata or pustular syphilides of the fold, extending to the bed and loosening its connection with the nail, and impairing the nutrition of the latter. Gummata may form on the periosteum beneath the bed, by which the bed and nail are raised up; if the gummous tumor breaks down, the ulceration readily extends to the bed and substance of the nail.

The fasciæ and tendons are often the seat of pain or of tenderness on pressure, especially if any eruptive fever attacks the patient. Severe pain sometimes occurs in the bursæ mucosæ and tendinous sheaths with which, in a few cases, inflammatory swelling is associated. Muscular and articular pains are also frequent; the latter sometimes associated with swelling, and closely resembling acute articular rheumatism.

Many patients suffer much in their bones, especially the tibia and skull. Sometimes there is deep-seated pain, hard to localize; sometimes there are well-defined spots of sensitiveness to pressure. There may be no further perceptible alteration; but in some cases there is inflammatory swelling of the perisosteum, easily detected by passing the finger lightly over the skin, or visible to the eyes as prominences, over which the skin may be slightly reddened, swollen, and hot. Many patients are plagued with boring pains (dolores osteocopi s. terebrantes), coming in the early part of the night and passing away with a gentle perspiration in the early morning hours.

The mucous membranes of the nose and the larynx are subject to erythematous, roseolar, and condylomatous disease. In the nose, the symptoms are a burning and itching, a feeling of dryness and pain, sometimes a bloody and fetid discharge; in the larynx, tickling and inclination to cough, and hoarseness. The exact nature of the alterations in each case can only be learned by rhinoscopic and laryngoscopic examination.

Syphilitic catarrh of the fauces may cause inflammation of the Eustachian tube, and injury to the hearing. Moos and Roosa have shown that internal periostitis of the petrous bone and disease of the labyrinth may also occur, and may weaken or destroy the power of hearing.

Disease of the eye often occurs at the end of the second stage, or as a transition to the third stage. Iritis is the most frequent affection; it is sometimes the ordinary form, without special peculiarities, and sometimes is gummous. In the latter case there are a number of small tumors on the iris, especially in the neighborhood of the pupil; sometimes yellowish, sometimes brownish in color. Next in frequency are affections of the choroid in various forms, including irido-choroiditis, serous choroiditis, and excessive development of pigment in patches (choroiditis pigmentosa disseminata, Hock). Many authorities refer disseminated choroiditis to syphilis, and regard it as a certain sign. Inflammation of the retina and optic nerve (retinitis, neuritis) also occur. Inflammatory

changes of the cornea sometimes occur, taking the form of gray spots of the size of pins' heads, and called keratitis punctata by Mauthner.

Severe nervous disturbances occur in many cases. Fournier showed that circumscribed anæsthesia is common, especially on the hands and forearms. Many complain of violent neuralgia. Facial palsy has been seen. In a case of my own, chorea appeared soon after an eruption of syphilitic roseola, but disappeared under a course of mercury. Many suffer from obstinate sleeplessness; others are greatly depressed.

If a rational treatment is pursued, tertiary symptoms may often be avoided, but not always. Many of the older authorities state that syphilis has no tertiary symptoms, but that what are called such are merely consequences of the use of mercury. But it not infrequently happens that patients present only the tertiary symptoms, showing no traces of the secondary ones, and never having had a course of mercury. They are often ignorant of any infection. Such patients sometimes evidently have no wish to deceive the physician. It follows that primary and secondary symptoms may give so little trouble that they are overlooked by the patient. No syphilitic person is sure, in spite of the best treatment, that he will not have tertiary symptoms; many have married, have had healthy children, have felt in good health for ten, twenty, even thirty years, but suddenly the stealthy foe bursts out from his ambush, and brings tertiary symptoms at a time when all seemed safe.

The broad condyloma being the characteristic form in secondary symptoms, the gumma or syphiloma leads among the tertiaries; whence Ricord proposed the name of gummous period. Special detail of symptoms will begin later on.

All tertiary symptoms lead to a protracted course, and often last for many years. One organic system after another may be attacked; scarcely are the symptoms conquered in one place when they break out anew in another. Injuries or bad habits sometimes determine the attack to a special organ: thus, persons who overstrain the mind are more liable to cerebral syphilis; drinkers, to that of the liver, and so on.

A final stage of marasmus is especially apt to come in neglected cases. In order to produce it, it is not necessary that there should have been tertiary symptoms, with suppuration and loss of fluids. The patient becomes pale, loses strength, keeps his bed, and at last dies of weakness, if help is not obtained in time,

Among the sequelæ are amyloid degeneration of almost any of the organs (which need not be preceded by suppuration), consumption, chronic nephritis, aneurisms, and psychopathies. Pigment-syphilis is described by Schwimmer as a formation of brown spots on the skin. Syphilitic leucoderma is a sort of contrast to this, appearing chiefly in women, on parts of the neck where there had been roseola or papules; it presents white patches, explained by Riehl by the fact that the pigment is carried from the deeper layers of epidermis into the cutis by wandering cells. I once saw hypertrophy of the entire skin, a sort of ichthyosis, in a man—keratosis syphilitica; it is not rare on the palms of the hands and soles of the feet, often combined with painful rhagades.

The most dreadful thing about this disease is that we can never be sure against relapses. They are often preceded by febrile movement and renewed enlargement of the spleen. Sometimes they are brought on by injuries. It is a fact that wounds often heal badly in persons with latent syphilis, and do not begin to cicatrize until antisyphilitic treatment has been adopted. Relapses after a thorough treatment, adopted perhaps at

the time of the first secondary symptoms, are almost the rule. They most frequently occur as mucous patches on the mucous membrane of the mouth and fauces. The relapses may occur at first every six or eight weeks, but by degrees they grow rarer and slighter, and in the second year usually cease entirely.

A question often put is that of marriage. Marriage should never be contracted previous to the end of the second year ; it is best to postpone until the end of the third year, and even then it must not be permitted unless relapses have been absent for at least six months past. Married men must look to their health, must be examined, if possible, by a good physician every fortnight or month, and must begin a specific treatment as soon as symptoms appear.

III. Diagnosis.—This may offer serious difficulties in all stages.

The soft chancre appears very soon after impure coitus—is often multiple, is painful to pressure and bleeds easily, suppurates freely, has no sharply defined hard base– and causes sympathetic buboes, usually of one side only; while the hard chancre is painful, and causes tenderness and inflammation of one, or a few only, of the lymphatic glands.

Many chancres begin as a distinct soft ulcer, and afterwards turn to the hard kind (mixed chancre). Patients with soft chancre must therefore be kept under observation for some time after their sore is healed.

An unusual situation—on the lips, fingers, eyelids, lobe of the ear, nipple, rectum—may mislead us as to the nature of a chancre. We should entertain suspicion if a source of infection has existed, and if subsequent wounds have been very hard to heal. Add the hard, well-defined base, sometimes raised like a wall, and above all the indolent multiple swelling of neighboring lymphatic glands, *e. g.*, under the chin, in lip chancre; above the internal condyle or in the axilla, in finger chancre; in the side of the thorax or axilla, in nipple chancre, etc. I know several instances of colleagues who contracted specific infection in their practice, and whose disease was long doubted, even when distinct secondary symptoms were present.

The recognition of secondary symptoms is especially hard when the primaries have disappeared, and when it is doubted, for instance, whether a certain exanthema is syphilitic or not.

Syphilitic exanthemata possess the following marks, as distinguished from the non-syphilitic:

a. They cause no itching.

b. They have a brown-red, coppery color, which is connected with the fact that many red blood-corpuscles leave the vessels *per diapedesin* at the affected parts of the skin, remain a long time, and undergo a gradual change of the coloring matter.

c. They often display polymorphism, that is, many kinds in combination; maculæ, papules, pustules, and squamæ succeed each other or stand side by side.

d. They tend to form groups; clusters, circles, or serpentine lines, rather than irregular arrangements.

e. They choose certain regions, as the boundary between forehead and hairy scalp, the soles and palms. Those on the front edge of the hairy scalp are called *corona veneris*, whatever their special character may be.

f. The action of remedies often gives a clue. Syphilitic exanthemata usually recede quickly when iodine and mercurials are given.

The syphilitic nature of chronic inflammation of the fauces, larynx,

and nose is often misunderstood, and resists all treatment until mercury or iodine relieves the patient. We very often have occasion to decide whether whitish or grayish spots on the mucous membrane of the mouth and fauces are syphilitic relapses or not. Smokers and persons with defective or pointed teeth often have such spots; persons who have had the mercurial inunction for syphilis, and irritated their mouths by frequent washing with chlorate of potash, may have similar appearances, not specific in their nature.

The diagnostic difficulties become almost insuperable when tertiary symptoms appear, and no previous history of syphilis can be obtained. The severest affections of the nervous system—chronic heart disease, pseudo-phthisical affections of the air passages, stricture of the œsophagus and rectum, disease of the liver and kidney, etc.—or chronic disease of bones, muscles, and joints, may be produced by tertiary syphilis. The importance of correct diagnosis in such cases is immense. After many years of fruitless treatment, a course of mercury or iodide of potassium may accomplish most brilliant results; in doubtful cases, these remedies should at least be tried.

Certain coarse anomalies may lead our attention to syphilis—thickly clustered white pigmented cicatrices on the forehead, sunken bridge of the nose, holes in the hard palate, cicatrices in the fauces, cicatrices and deformities of the epiglottis, or cicatrices on the legs, genitals, and anus. Mydriasis of one eye, and paralysis of the eye muscles, originating without demonstrable injury, may excite suspicions of syphilis.

Repeated abortions of married women may be held to be suspicious; syphilis is generally the cause, usually that of the father.

IV. Prognosis.—In acquired syphilis, this is so far favorable, as that surprisingly favorable results are obtained more quickly and surely than in most other diseases, if we understand the nature of the complaint. Neglect, however, may result in irremediable damage.

But we can never be sure that the disease is permanently cured. Relapses are very frequent; tertiary disease of internal organs may prove fatal, in spite of all care.

Accidentally acquired syphilis is, in general, very obstinate and malignant, as physicians have often found to their sorrow. This may be due to the fact that the disease is overlooked, and not treated until a late period.

Cases acquired by coitus with foreign races are considered especially dangerous; the Chinese form is dreaded by sailors. The stage in which the disease is has its weight in the prognosis; all tertiary symptoms are the most dangerous, as they compromise vital organs.

V. Treatment.—In respect to prophylaxis, the principles stated under gonorrhœa (Vol. IV., p. 249) apply. To prevent accidental contagion, disinfect instruments carefully; examine wet-nurses for marks of syphilis, and always about the arms and genitals; use for the supply of lymph in vaccination only well children older than six months; employ lymph that has no tint of blood, etc. After an impure connection, there is little use in washing with carbolic acid, vinegar, and the like. Even deep cauterization of a fresh erosion with nitrate of silver or caustic potash has been found incompetent to prevent the development of induration and further symptoms of syphilis.

For existing syphilis, every physician has his own treatment; we will state the plan which has been serviceable to us in a very large number of cases.

For hard chancre, order two and a half drachms of emplastrum mercuriale, to be spread on the sticky side of adhesive plaster in a layer as thick as the back of a knife, reserving a border all around for the plaster to adhere by. A light bandage may be added. The plaster is to be of a shape and size to cover the sore and extend a little beyond the edges. In ulcers on the glans or inner surface of the prepuce, insert the dressing, with the ointment towards the sore, inside of the prepuce. The plaster must be renewed morning and evening, and continued until the ulcer has become completely soft. Open ulcers do not contra-indicate this treatment; they heal remarkably quickly under it. The hard lumps usually soften and disappear very soon. Softening is not sufficient; there must be complete disappearance to avoid the danger of relapse as far as possible. If the spot once treated should again become hard, it must be treated again in the same way.

It is much disputed whether general treatment is applicable for the primary symptoms. It has been said that this only retards the secondary symptoms, and makes the disease a lingering one. We do not at all agree with this, considering the hard chancre as by no means an exclusively local disease, but as indicating the accomplishment of general infection, since several weeks after infection are required to produce it, which is long enough for general infection. Hence the adoption of general treatment seems to us entirely justified. We have repeatedly observed cases in which, the treatment being carried out, no secondary symptoms at all occurred, or those that appeared were unusually mild. We advise the use of an inunction cure with mercurial ointment (ungt. hydrarg. cinereum, one drachm for each daily treatment).

If possible, a daily bath is taken, after which the above quantity of blue ointment is rubbed in upon the leg; on the following days the application is changed successively to the following parts: the thigh, then leg and thigh of the other side, then upper arm of one side and the other, breast, abdomen, and back again to the leg. The part rubbed must always be carefully washed on the next day, before the new part is touched. If a bath is inadmissible, lukewarm water and soft soap are used. When thirty doses are used, we may leave off, if there are no syphilitic symptoms.

The manner of inunction is very important. Let the patient take the lump in the hollow of his hand and rub it slowly back and forth over the skin until the latter is no longer sticky and greasy, but is dry. Many health resorts, justly renowned for curing syphilis, owe their reputation, not to the waters, but to the skill of the rubbers. Hairy places are to be avoided, if possible; a hairy breast or abdomen, for example. Neglect of this rule easily causes inflammation of hair-follicles, which protrude as nodules, lumps, and pus-bladders, making the so-called eczema mercuriale. This is no great harm, for the eruption heals spontaneously in a few days. The most strict and careful directions are not superfluous; we must, especially at the beginning, make sure by the eye and the touch that the rules are observed.

While the inunction cure is in progress the patient is to gargle after each meal with chlorate of potash (1 : 20).

Patients must not smoke; they must keep the teeth clean, especially if any are defective. Prescribe for this purpose: ℞ Ossium sepiæ pulv., ʒ iss.; Magnesiæ carb., Sapon. medic., āā ʒ iiss.; Ol. menth. pip., gtt. v. M. S. Tooth-powder.

All these precautions are intended to prevent mercurial stomatitis.

Many patients complain of a disagreeable metallic taste in the mouth before the stomatitis appears; then there is salivation; the gums swell and grow soft, the teeth feel loose, and in fact they are so; epithelium is thrown off and ulcers form; the breath is fetid, and adhesions may occur between the cheek and tongue which will be very hard to remove by a subsequent operation. The cheek is especially apt to be sore where the crown of a molar tooth touches it. Many persons are so sensitive to mercurials that a much smaller dose than one drachm produces mercurialism.

In addition to mercurial eczema, ptyalism, and stomatitis, albuminuria may result from mercurial inunction. I have seen this twice within the past year in women; Fürbringer states that he has observed it in eight per cent of his patients. The amount of albumin is usually small. The sediment is often wanting. Suspension of treatment arrests the trouble more or less rapidly. Mercurial diarrhœa sometimes supervenes, and may take a dysenteric form.

In addition to the medical treatment, we must not forget diet. The food should be non-irritating, but strengthening; the hunger-cure is not needed. Excesses in wine and women must be avoided; it is not right to place others in danger of infection. The patient may go out daily, taking all pains to avoid dampness and taking cold, to which the mercurial treatment renders one subject. Cold baths and douches are to be avoided; woollen underclothes are to be worn in winter, to avoid colds as far as possible.

During the secondary period, the medicine and diet remain at first the same as above. Many direct the use of iodide of potash in addition to the inunction (five per cent solution, one tablespoonful three times a day), but we do not much like this combination, having often seen an abundant and unpleasant eruption of furuncles follow it. We never found that the symptoms of syphilis receded more rapidly, or were longer in relapsing, under this method.

Broad condylomata of the skin can be removed very quickly by sprinkling with common salt, and just afterwards with powdered calomel. Where two surfaces of skin are in apposition, insert cotton batting, to prevent friction. Excesses of all sorts are to be avoided for some time after all symptoms have disappeared. The patients are to be warned not to think themselves permanently cured, but to pay strict attention to themselves, and seek medical assistance at once when anything suspious is noticed. They are to be expressly told that relapses are the rule within the first two years.

In case of a relapse affecting both skin and mucous membrane, it is well to repeat the former treatment. If there are only condylomata of the mouth and throat, internal mercurial treatment usually suffices. We prefer the yellow iodide, and, to avoid diarrhœa and pain, we like to combine with opium. (℞ Hydrag. iodidi flav., gr. viij.; Opii, gr. v.; Pulv. et succ. glycyrrhizæ, q. s. ut ft. pil. no. 30. S. Three times a day, one pill after eating.)

During summer, baths of brine, iodine, and sulphur are useful.

While mercury is one of the most valuable remedies in primary and secondary syphilis, iodine preparations are of special use in the tertiary stages, especially iodide of potassium (five per cent solution, one tablespoonful three times a day). Mercurials, however, are not superseded; they often act very quickly. Gummous ulcers of the skin often heal very

quickly under the influence of mercurial plaster; chronic bone disease improves surprisingly during inunction, etc.

Iodine preparations, especially with iron, are the chief remedies for syphilitic cachexia and amyloid degeneration: *e. g.*, ℞ Potass. iod., Ferri lact., āā ʒ iiss.; Quininæ hydrochl., gr. xv.; Pulv. et succ. glycyrrh., q. s. ut f. pil. no. 100. S. Three times a day, three pills, after eating.

Baths of brine, iodine, sulphur, are very useful at this period.

It may be useful to notice some of the many treatments for syphilis.

Excision of the hard chancre has been repeatedly tried, in the hope of preventing the development of further symptoms. The incision must be made in sound tissue, far beyond the diseased part. The results are reported variously, but few can claim to have successfully nipped syphilis in the bud. The cicatrix is usually found to become indurated and form a second, but larger, hard chancre; or, if the excision seems to be successful, secondary symptoms nevertheless follow. In two cases of our own, we failed entirely; nor can we justify the operation, believing that hard chancre is the first evidence of general infection.

There is much controversy about the use of quicksilver and the different preparations. Subcutaneous injections, fumigations, baths, and suppositories have been recommended.

For subcutaneous injection, corrosive sublimate has been much used, and has been supposed to give the speediest and most lasting results. Lewin has given it the most careful test. Charles Hunter (1856), Hebra (1860), and Scareno (1865) first used subcutaneous injections of mercurials. A silver canula is required. The best spots are the back and sides of the thorax; abscesses are easily produced in other parts. The best care will not always prevent abscesses; the injection is very painful; gangrene and death have once occurred. To lessen the pain and inflammation, J. Müller directed the addition of ten parts of common salt to one of the mercurial. Bamberger used an albuminate of mercury, and afterwards a peptone, for his subcutaneous injections, while Sigmund recommended bicyanuret of mercury; Liebreich, formamid of mercury; Wolff, glycocoll, alonin, and asparagin, in combination with mercury, and Schütz a solution of hydrargyrochloride-urea. Calomel suspended in water has also been tried, but has caused inflammation and abscesses.

Calomel is used for fumigation. The patient is placed on a chair, an alcohol lamp is put under the chair, and over the lamp a tin stand with powdered calomel; the patient is wrapped in blankets to the shoulders.

Sublimate is used for baths (ʒ iiss. to a bath).

Suppositories of blue ointment have been introduced into the rectum; but Fürbringer shows that mercury is very poorly absorbed by that part.

Among internal preparations, the iodide (already mentioned), chloride, bichloride, and biniodide are used.

Corrosive sublimate is the basis of many unduly praised systems of treatment, of which Dzondis' is the best known. It irritates the stomach, cannot be given in solution or powder, and must always be taken on a full stomach. (℞ Hydr. chlor. corr., gr. iss.; Pulv. et succ. glycyrrh., q. s. ut f. pil. no. xxx. S. Three times a day, one pill, after eating.)

Calomel is unsuited for continued use, as it irritates the stomach and intestine. It may be given in powder or pill, one-half grain twice a day. The biniodide also has irritating effects upon the mucous membrane of the intestine. (℞ Hydr. iodid. rubri, gr. iss.; Pulv. et succ. glycyr., q. s. ut f. pil. no. xxx. S. Three times a day, one pill.)

Blue ointment and regulus of mercury have been given in the form of pill.

Many physicians strenuously oppose the giving of quicksilver in any form for syphilis. Such "cranks" recommend also the treatment by hunger, purgation, sweating, and particularly decoctions of woods. Guaiac, sarsaparilla root, and sassafras are used this way. Sarsaparilla forms a chief component of Zittmann's decoction, which is made of two strengths, the patient taking one-half to one pint of the stronger in bed in the morning, and as much in the evening of the weaker; the diet to be small in amount and poor in albumen.

Many attempts have been made to replace mercury by other metals—gold, silver, platinum, copper, arsenic.

Iodide of sodium or ammonium have been proposed instead of iodide of potassium, but we are not satisfied of their superior effect.

Many other remedial measures are required at different times, which may in part be found in text-books on surgery, ophthalmology, and aural diseases.

2. *Tertiary Syphilis of the Skin, Muscles, Fasciæ, Joints, and Bones.*

I. Symptoms and Diagnosis.—Gummata of the skin and subcutaneous tissue are either felt or seen as lumpy projections, from the size of a pea to that of an apple. The skin over them is often blue-red, shiny, and thin. They are especially common on the leg below the knee. They often soften and burst outward. The fluid contents usually dry into brown or gray-green crusts on the surface. Successive portions thus dry up, forming a sort of layers, the lower ones being broader than those above them, and forming crusts like an oyster-shell. The form has also been compared to that of cows' dung. The name of rupia (or rhypia) syphilitica has been given to it. On raising these crusts, we find an ulcer of considerable depth, with step edges, and a greasy, tallowy coating—striking points which can be of use in distinguishing these from similar non-syphilitic eruptions. The ulcers often heal at one point and spread at another. This finally gives rise to peculiar shapes like that of a kidney or horse-shoe; the cicatrices which follow are colored brown-red, and when old are white with a brown edge—appearances partially characteristic of syphilis. Cutaneous gumata sometimes lie so close together that when they heal the skin is traversed by numerous cicatrices which may contract and cause distortion. Ulcerating gummata may also cause deformity of the alæ nasi, ears, lips, etc. Gummous tumors seldom become carcinomatous. They often undergo slow spontaneous absorption, leaving places where the skin is sunken and thin. Gummata of many other organs are often associated with those of the skin; the process often lasts many years. Many years have usually elapsed from the first infection to the development of gummata of the skin.

The muscles may undergo important changes—sometimes diffuse syphilitic infiltration of the interstitial connective tissue, with fatty change, and disappearance of the muscular substance proper, sometimes the development of circumscribed nodes of gumma, which at times suppurate or become absorbed and leave cicatrices of connective tissue, or adhesions to neighboring organs. Gummata often consist of deposits inside of diffuse infiltrations. The clinical signs are stiffness, pain, and contracture of the muscles affected; the lumps and scars may be felt and sometimes seen. Over-exertion of certain groups of muscles, or injuries, may cause the local development of syphilis. One muscle alone may be affected or several at once.

Keyes mentions the not infrequent swelling of bursæ mucosæ, as a

cause of which he assumes gummous tumors, though without supporting his view by autopsies. Slight injuries often form the starting-point of the affection.

The sheaths of tendons have been the seat of inflammatory swelling or circumscribed swellings—hygromata syphilitica—in a few cases. The fasciæ may also become the seat of gummata.

Joint disease is often connected with syphilis. Gummous and inflammatory processes near the articular ends of bones may involve the joints in the affection; or else gummous knots may develop in the subserous tissue of joints, causing pain, swelling, impairment of function, or even suppuration and ankylosis. Symptoms of chronic hydarthrosis may appear; in other cases, symptoms resembling those of acute rheumatism may occur very early.

The bones are often affected by tertiary syphilis, but the affection

Fig. 62.

Ulcerating gummata of the cranial bones in a woman aged 43. Author's observation. Zurich clinic.

may occur so early as to form part of the first symptoms. Gummata form upon them, proceeding from the periosteum or the medulla. In the former case, there are firm soft prominences, selecting as their chief seat the bones nearest to the skin, as the skull, clavicle, breast-bone, ribs, shoulder-blade, anterior edge of the tibia, fibula, ulna, etc. These gummata follow the course of the blood-vessels into the bones, where they form polypoid and root-shaped processes. The portions between them disappear by degrees till patches of bone are completely gone. Gummata proceeding from the medulla may in like manner cause gradual thinning of the bone-substance, so that very slight external causes sometimes produce fracture. Gummata of the bones sometimes soften and burst externally, causing further destruction of the bone-substance. The scalp sometimes seems punched out, as in Fig. 62, laying bare the white carious worm-eaten bones of the skull. Thick pieces of the bone are even separated, and the pulsating meninges are seen. Caseous and calcareous alterations sometimes occurs in gummata. If gummata are

caused to diasppear by antisyphilitic treatment, depressions of the bones often remain in their place, to which the thinned skin seems to be adherent.

Ostitis and periostitis sometimes occur in syphilitic patients, not originating in the breaking down of gumma nodes, but of a more general character. Periostitis usually begins in the inner layers of the periosteum, and forms at first an infiltration which may be pitted by pressure, but afterwards ossifies and forms a hard prominence—tophus syphiliticus. It is doubtful whether the latter may originate in the ossification of gummata. The attachments, or even whole muscles, may be ossified, causing myositis deformans.

In other cases, periostitis causes the formation of pus, abscesses, or fistulæ; or gradual absorption takes place. Dactylitis syphilitica, a disease of the phalanges of the fingers, often causes considerable swellings—spina ventosa syphilitica.

Spontaneous fracture has often been seen in syphilitic patients; syphilis, like many chronic diseases, leading to atrophy and fragility of bone—osteopsatyrosis.

Bone syphilis is one of the most dreaded forms. It tortures the patient with pain, sometimes lasts during the whole of life, causes marked and permanent deformity, and often renders a patient unable to labor. Danger to life is less imminent; but long suppuration of the bones may lead to amyloidosis, and extensive syphilis may involve the cranium, meninges, and brain. It is not always easy to recognize the disease; it is liable to be confounded, for instance, with tuberculous processes.

Bone syphilis is one of those diseases which have often been said to be due, not to syphilis, but to mercurials. This view is refuted by the fact that the disease occurs in patients who have never used mercurials. Yet this drug certainly has great influence on the bony system. Trustworthy authorities have reported the finding of globules of mercury in the bones of patients treated with the remedy; and congestive changes in the medulla of bones are known to occur under the influence of mercurials. Nor can it be denied that the long-continued action of mercury may cause necrosis of the jaw and great brittleness of bones.

II. Treatment.—Among the internal remedies, we have preparations of iodine; and among the external, ointments of iodine and mercury, mercurial plaster, baths of iodine, brine, and sulphur. Ulcerative processes are often found to disappear with surprising rapidity under the influence of the plaster. Surgical interference may be needed: necrotomy or trepanation; and in general, syphilitic disease of bone or joint belongs to surgery rather than to internal medicine.

3. *Syphilis of the Nose.*

I. Symptoms and Diagnosis.—The hard ulcer on the outside of the nose is usually on the tip or the alæ, but is rare.

The mucous membrane very often shares in the changes of the second period. There may be diffuse erythema, or hyperæmia in patches, or broad condylomata, which break down, and sometimes lead to necrosis and loss of substance in the mucous membrane, cartilage, and bone. Burning, dryness, heat in the nose, stopping-up of the passages, abundant, sometimes ill-smelling discharge from the nose, subjective perception of bad smells, and sometimes ozæna, often accompany the condition

described; the causes for which can be easily discovered with the rhinoscope.

Gummous disease of the nose usually belongs to the later stages; it sometimes proceeds from the outer skin, sometimes from the mucous membrane, or from the cartilages or bones. The breaking down of gummata causes loss of skin, easily confounded with tuberculosis of the skin (lupus), sometimes with cancer, and leaving unpleasant deformities of the nose after cicatrization. Gummata on the mucous membrane often render the passage impervious, or during decay cause a stinking discharge, or involve the cartilage or bone. Gummata of the cartilages are not common. If they break down, a hole or fissure connecting the two nostrils may be formed, without much subsequent damage; or, if the damage is extensive, the front of the end of the nose sinks in, and both nostrils form one hole. Gummata of the nasal bones may develop on parts of the bony frame of the nose. If the septum is involved, and the destruction is extensive, the bridge of the nose may sink in like a saddle, so that the existence of prolonged syphilis can be read in the face. Similar deformities, however, occur after fracture of the bony ridge of the nose. Gummous destruction of the ethmoid bone often causes loss of the nerve ofsmell and permanent anosmia. If the cribriform plate is attacked, there is danger of meningitis, and perhaps death. The floor of the nostrils is often attacked by ulcerous gummatous processes, causing destruction, and opening connections between mouth and nose. Such communications are very characteristic of previous syphilis, and easily lead to disturbance in eating or drinking, or to changes in the voice, which acquires a nasal and indistinct character. It is remarkable how slight a discomfort often attends these processes. I have repeatedly attended patients who had scarcely had any pain, yet the bony ridge of the nose was a yielding, crepitant mass. In case of perforation inwardly, I have seen emphysema of the skin. A bad-smelling discharge—ozæna syphilitica—is often kept up by ulceration of bone. The patients often blow out pieces of bone. Figure 63 represents a part of a thin lamella of the concha, which a patient of mine discharged while blowing his nose; but the entire concha has been blown away in this manner. Gummous processes are usually easy to recognize rhinoscopically. Examination with the sound often discovers bare rough bone.

FIG. 63.

Piece of bone discharged spontaneously in syphilis of the nose. Natural size. Author's observation.

Syphilitic ozæna does not always depend on ulcerative processes of the nose; syphilis, independently of the latter, may lead to chronic inflammation of the mucous membrane of the nose—rhinitis syph. atrophica—with atrophy and retraction, which is associated with obstinate ozæna.

II. TREATMENT.—Besides the constitutional use of iodine and mercury, local treatment is very important. For ulcerations of the outer skin, we recommend mercurial plaster. For destruction of the inner surfaces, the nasal douche; using carbolic acid (two per cent), sublimate (one-tenth per cent), or thymol, followed by pencilling with ℞ Potassii iod., gr. xv.; Iod., gr. iss.; glycerini, fl. ʒ ij., or a snuff powder of equal parts of calomel and alum, or iodoform. Surgical operations are included, as necrotomy, or plastic operations after the disease is arrested and healed.

4. *Syphilis of the Larynx.*

I. Etiology.—The larynx is often affected in syphilis. A frequent use of the laryngoscope, not confined to periods when it seems to be demanded by active symptoms, will show the presence of slight affections oftener than is usually stated to be the case.

A very variable period may elapse between the initial symptoms of syphilis and those of the laryngeal trouble. The latter may appear among the first of the secondary symptoms—from six to eight weeks after the hard chancre—but years may elapse (according to Türck, thirty years) before the larynx is attacked.

The outbreak of syphilitic changes may be favored by accidental injuries to the larynx, including colds, or very loud speaking. I have found syphilitic symptoms of the larynx frequent in great smokers.

During childhood, the disease is rare; it occurs in new-born infants, however. Rauchfuss says that there is no distinction between hereditary and acquired syphilis in the form of the laryngeal disease. It is said, however, that laryngeal syphilis has a more rapid course, with a greater tendency to ulceration, in children than in adults.

II. Symptoms and Anatomical Changes.—The chief symptoms are recognized by the aid of the laryngoscope, which enables us to study the living anatomy.

Distinguish strictly between primary and consecutive changes. The primary include syphilitic catarrh, broad condylomata of the mucous membrane, and gummata.

Many refer chorditis vocalis hypertrophica inferior (see Vol. I., p. 181) to syphilis.

In many cases, the above-named primary changes cause ulcerations, which may lead to very extensive destruction in the larynx, or to œdema and laryngeal perichondritis, or to adhesions, cicatrices, and stenosis of the larynx. The great danger usually lies in the consecutive, rather than in the primary disease.

Syphilitic catarrh of the larynx has no peculiarities to distinguish it from the non-syphilitic form, when examined by the laryngoscope alone. Abnormal redness, swelling, and increased secretion are its chief symptoms. The swelling of the mucous membrane may be so great as to produce symptoms of stenosis.

It may be acute or chronic; if the true vocal cords are affected, it causes hoarseness, which may be of itself a suspicious fact. If the catarrh does not impair the vocal vibrations, the voice is not injured; and the disease is not recognized unless we make a rule to use the laryngoscope, even when no symptoms seem to require it. Subjective troubles may be entirely absent; there is often complaint of tickling, slight burning in the larynx, and disposition to cough.

Many syphilitics, especially those of the anxious type, complain of these symptoms, although not the least change can be discovered by external or internal examination. There is here a sort of hyperæsthesia of the mucous membrane, maintained by a hypochondriacal disposition.

If syphilitic catarrh of the larynx appears at the very beginning of the secondary stage of syphilis, it may be placed side by side with angina; it often seems to be but an extension of the latter downwards. In other cases, it occurs much later, or accompanies a relapse, or, rarely,

it forms the only relapse of syphilis. If the intensity is great or the duration long, it causes ulceration. The ulcers are usually small and most commonly situated on the true vocal cords and arytenoid cartilages.

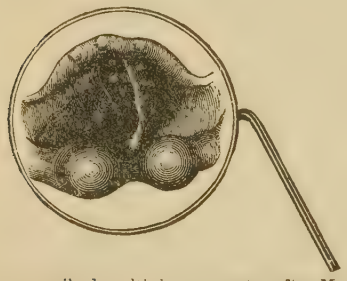
Broad condyloma of the mucous membrane of the larynx consists of white elevations, most frequently seated on the true vocal cords, next on the arytenoid cartilages, posterior wall of the larynx, and ary-epiglottidean folds. The elevations look like the broad condylomata of the pharyngeal mucous membrane.

If proper treatment is not given, the surface ulcerates, and the ulceration may spread.

Gerhardt and Roth have given the first thorough description of condyloma of the laryngeal mucous membrane. Some (Lewin, Waldenburg) incorrectly deny the existence of broad condyloma in the larynx.

Türck gives two cases in which pointed condylomata appeared on the mucous membrane of the larynx of syphilitic patients and disappeared when treated mercurially, though the pointed condyloma has no direct relation to syphilis. Papillary excrescences, sometimes formed on the edge of syphilitic ulcerations, must not be confounded with pointed condylomata.

FIG. 64.



Circumscribed multiple gummata, after Mandl.

FIG. 65.

Syphilitic stenosis of the larynx, due to the formation of a diaphragm between the two vocal cords. Author's observation.

Gummous syphilis of the larynx is a late symptom. The tumors are sometimes distinct and circumscribed, and have the size of a pin's head, a pea, or more; at other times they form a diffuse nodular infiltration of the mucous membrane (see Fig. 64). In either case they may cause symptoms of stenosis. The favorite seat is the epiglottis; the disease often passes directly from the fauces to the epiglottis. This organ is often converted into an irregular globular or roller-shaped mass which gives no trouble, and may even be discovered accidentally at the laryngoscopic examination, as I have twice found in men who applied for a certificate for life insurance. Gummata may occur on the vocal cords and other parts of the mucous membrane of the larynx.

Several clinical stages may be distinguished. Schech names four: that of infiltration, of softening, of destruction, and of absorption. When a gumma begins to ulcerate, great devastation may occur; cicatrization may take place on one side while ulceration extends on another, forming a serpiginous ulcer. The ulcerative process is usually chronic, but Frankl has described the case of a new-born child in whom barely three weeks elapsed from the first hereditary symptoms in the larynx to death.

The chief danger lies in the consecutive changes, and increases with

every delay in treatment. The first consecutive changes are the ulcerative process and its results.

Dangerous bleeding is rarely caused by ulceration, though Türck has published a case.

In many cases, ulceration causes œdema of the glottis; or extends to the perichondrium and involves danger of perichondritis laryngea. Loss of some of the parts of the larynx—for instance, the entire epiglottis—may occur.

Loss of the epiglottis does not always cause trouble in swallowing. The false cords may lie so close together during the act as to prevent food from entering; and the stylo-glossus may draw the tongue backward so as to cover the entrance to the larynx.

Danger is not always past, even when the ulcers are cured; for neighboring parts often adhere, and may lie over the glottis like a diaphragm, causing progressive stenosis (see Fig. 65). Or, the increasing retraction of the cicatrix may lead to such dislocation and impairment of movement as to cause permanent injury to function. The several parts can sometimes hardly be recognized with the laryngoscope.

III. Diagnosis.—If the patient is known to have had syphilis, or if he bears the marks of it on other parts of the body, the diagnosis is easy. The points to notice are eruptions, swelling of the lymphatics, cicatrices of the genitals, recent or cicatrized disease of the pharynx, etc.

If patients deny syphilitic infection (and *mendacia syphilitica* is almost proverbial), and if indications are absent, the diagnosis may be difficult in case of ulcerative processes. It may be impossible to distinguish tuberculous ulceration, unless the bacilli of tubercle can be shown. Josset-Moure has paid special attention to this point of diagnosis. It is a very important fact that syphilitic affections of the larynx are mostly painless, and are usually associated with swelling of the cervical lymphatic glands, which is not the case in phthisis. Disease of the lungs is also an important element to consider. In lupus and lepra, there is usually the same affection of the skin present at the same time. Cancer of the larynx offers more difficulties, but the breaking down of the tumor takes place more rapidly, and causes great pain.

Phthisis and syphilis of the larynx are sometimes combined, in which case it is impossible to draw a line between the two.

IV. Prognosis.—This is always serious, for, though it is often possible to control the processes, yet the residua are often not free from considerable danger. The result will be better, in proportion as the disease is treated in season. Stenosis is especially serious, because it is hard to reach by treatment, and is inclined to grow worse; it often compels the patient to wear a canula in the trachea all his life.

V. Treatment.—This includes prophylactic, the general, and the local remedies.

Patients must be as careful as possible of their larynx, avoiding colds, continued loud speaking, and excessive smoking.

If the laryngeal disease is recognized as syphilitic, general specific treatment must be ordered at once.

The local treatment may include (for catarrh) inhalations of a weak solution of sublimate (gr. $\frac{1}{3}$–$\frac{1}{2}$: ℥ vi.). For condylomata of the mucous membrane, insufflation of calomel into the larynx (Calomel, gr. xxx.; Pulveris acaciæ, ʒ ij., once a day). For gummata and ulcerous processes, paint the interior once a day with iodide of potash, gr. xv.; iodine, gr.

iss.; glycerin, fl. ʒ ij.; or with dilute tincture of iodine; or let dilute solution of iodide of potash be inhaled (gr. viij.–xv. : fl. ʒ vi.).

Complications and sequelæ are partly subjects for surgical treatment.

5. *Syphilis of the Trachea and Bronchi.*

I. Symptoms and Diagnosis.—Syphilis of the bronchi and trachea is much rarer than that of the larynx, but is otherwise identical. The larynx is sometimes affected at the same time, the process having extended thence to the upper part of the trachea; or the latter may have been originally attacked and the disease spread into the bronchi; or the disease extends through the entire course of the air passages.

At an early period in general syphilis, catarrhal (erythematous) alterations of the mucous membrane may occur; at least, we often hear syphilitic patients complain of tickling, tendency to cough, and expectoration, which are relieved remarkably soon by anti-syphilitic treatment.

Broad condyloma of the mucous coat of the trachea was first seen by Seidel with the laryngoscope, and afterwards confirmed by Mackenzie.

At later periods, knots of gumma may occur, oftener as diffused infiltration than as circumscribed tumors. They often come on insidiously, causing no symptoms but those of contraction of the passage; but in the act of breaking down and cicatrizing, they produce trouble and danger of many sorts. Purulent masses are expectorated with coughing. The secretion flowing downward may produce foreign-body pneumonia. Wilkis and Kelly saw the aorta and pulmonary artery attacked by ulceration, with fatal results. In other cases, the mediastinum is attacked by ulceration and destruction. Necrosis of cartilage often takes place, and the pieces that are coughed up threaten suffocation in their passage. Cicatrization brings the danger of stenosis of the trachea or bronchi. But the process is not always alike; growths like septa sometimes form, as in the larynx; at other times there are flexions, and cicatricial wrinklings, or the trachea sinks together. In case of stenosis, the larynx makes shorter excursions, not usually exceeding one centimetre; and the head is usually held forward; both of which symptons contrast with what occurs in stenosis of the larynx. For clinical symptoms of bronchial stenosis, see Vol. I., p. 244. The greater the longitudinal extent that is affected by stenosis, the more trouble and danger, even though the stenosis may not be very considerable.

Swellings in the neighborhood of the trachea have been found which receded when treated specifically, so that we may speak of syphilitic peritracheitis. The danger consists in compression of various organs, especially the trachea, or suppuration and destruction. Perichorditis of the bronchi seems to be similar; and the growth of connective tissue may extend into the lung-tissue.

II. Treatment the same as in syphilis of the larynx.

6. *Syphilis of the Lungs.*

(*Syphilitic phthisis.*)

I. Anatomical Changes.—It was affirmed by the older physicians that syphilis was the cause of certain forms of consumption. This view

has been especially sustained by Virchow in later times. The clinical difficulty lies in the fact that we are often forced to make the diagnosis of syphilis *ex juvantibus*, and the diagnosis may have subsequent doubt thrown on it. Of late years, many new cases have been added, and it seems as if the obscure relations between the two diseases would be cleared up.

Lung-syphilis may appear anatomically in two forms, a diffuse infiltration or circumscribed gumma-nodes. The syphilitic infiltration of the lungs prefers the middle part of the lungs; in Grandidier's experience, the middle lobe of the right lung is most frequently attacked. Among his thirty cases, twenty-seven affected that part, one the left lung, and three the apex. The latter circumstance is of special importance, as standing in opposition to the localization of tubercular consumption. The altered part is empty of air, feels firm, is dry when cut, and has a gray or gray-yellow or gray-white color, like a lung in fibrinous pneumonia in the stage of gray hepatization. Lorain and Robin described these changes in the lungs of syphilitic new-born children as epithelioma; they have often been termed white hepatization.

Greenfield found by microscopic examination that the chief process is interstitial proliferation in the lungs. The epithelium of the alveoli shows proliferation in many places, but may remain entirely unchanged in other respects. In other cases, it has undergone fatty or myelinic degeneration. Pancritius, the author of a recent monogram on lung-syphilis, considers the process as interstitial proliferation, always proceeding from the hilus of the lung. Cornil describes a peculiar change of the lymphatics of the lungs consequent upon syphilis, in which the epithelial cells increase and accumulate, and the lymph-corpuscles in the tubes undergo cheesy metamorphosis.

If infiltrations liquefy to pus, cavities form, and the clinical symptoms closely resemble those of tuberculosis. Cicatricial or cirrhotic changes, however, are also possible during resorption.

The gummous disease of the lungs differs from the above in assuming the form of circumscribed tumors. Softening may occur. Köbner describes a syphilitic new-born child which died with symptoms of pyo-pneumothorax, caused by a gummous tumor under the pulmonary pleura which afterwards broke down. In favorable cases, resorption and cicatrization may occur.

Virchow mentions a third form of syphilitic lung disease which externally resembles the brown induration of the lungs, as found in heart disease. Many have connected such changes as those of broncho-pneumonia and gelatinous infiltration with syphilis.

The pleura is usually infiltrated secondarily; it seldom appears to be affected independently of the lungs; cicatricial thickening and contraction are a prominent feature (pleuritis deformans syphilitica).

II. Symptoms.—Clinical symptoms of lung-syphilis may be absent, though extensive lung-disease exists. Moxon reports a case in which a person who had been syphilitic died suddenly in consequence of fracture of a vertebra, and at the autopsy there was found extensive infiltration in the left lower lobe, and scattered spots in the right lung without any symptoms during life. Meschede also describes a case in which gummata were unexpectedly found in the lung of an adult who had been infected with syphilis many years before.

The disease may express itself by symptoms like those of commencing tuberculous consumption. Langerhans gives the case of a young man

who had symptoms of catarrh of the right apex, with hæmoptysis; he had had impure coitus some time before; there was no hereditary syphilis in the family. A long residence in a southern climate did no good; several copious bleedings occurred. A complete cure was obtained by treatment with injections of sublimate. I saw an officer some months ago who had been sent to Davos, for condensation of the lower half of the left upper lobe, as a consumptive patient. There was no expectoration. Slight febrile movement in the evening. Emaciation progressive. Syphilitic infection some years before. Swelling of the clavicle and the right elbow, in the third month of his residence at Davos. Mercurial inunctions quickly caused the bone-disease to disappear. The fever disappeared in a week. Great appetite and rapid gain in weight. Healthy complexion. Complete recovery.

A third series of cases present the clinical symptoms of advanced consumption; those of infiltration being sometimes prominent, those of cavities at other times; but the sputa contain no tubercle bacilli.

III. Diagnosis, Prognosis, Treatment.—The diagnosis is not easy; one must have the divining power given by experience. We should always be on the watch if syphilitic members of healthy families have lung symptoms, for the prognosis is not very unfavorable if the cause is recognized quite early. The treatment includes the use of mercurials and iodine preparations.

Appendix.—Diffuse or circumscribed gummata are seen in the mammæ, which are dispersed by pencilling with iodine, rubbing in ointment of iodine or mercury, and the internal use of iodine and mercury.

7. *Syphilis of the Digestive Tract.*

I. Symptoms.—In the early stages of syphilis, the mucous membrane of the mouth is often the seat of erythematous and condylomatous changes which often lead to ulceration and loss of substance.

The same is seen on the tongue, occasionally roseolar spots. At a later date, gummous nodes often appear on the tongue; as distinguished from cancerous knots (which they resemble), they are solitary, not painful, and do not (like cancer) cause hard swelling of the nearest lymphatic glands. If early and suitable vigorous treatment is given, they may usually be dispersed, otherwise they soften and suppurate for a long time. Langenbeck and Hutchinson observed a transformation to cancer.

It was formerly believed that numerous depressions and incurvations of the surface of the tongue were due to syphilis. This view is as little justified as that, that thickening of the epithelial layer of the tongue, giving a thick and almost horny covering (psoriasis linguæ), is connected with syphilis. Both conditions may be found in inveterate smokers.

Gummata sometimes appear on the organs of the throat (see Vol. IV., p. 334, for early symptoms). If they break down, the uvula may be destroyed; extensive destruction of the tonsils and soft palate occurs, and the latter may adhere to the posterior wall of the pharynx, making a sort of diaphragm between the naso-pharyngeal cavity and the parts below, forming at last complete closure. Swallowing and speech are much interfered with by this change, besides which, abnormal adhesions and closure of this region are hard to remedy. The condition is equally unpleasant when such a diaphragm forms in the lower fauces. Large vessels, even the internal carotid, or the vertebral artery, may be found, and fatal bleeding result.

If gummata proceed from the submucous or periosteal tissue of the hard palate, they are first perceived by the firm soft swelling, and sometimes by increased redness of the mucous membrane. In case of suppuration and opening, abnormal communication between the mouth and nose is often formed, causing disturbance in eating, drinking, and speaking.

Cases of gummous tumors in the salivary glands are described.

Gummata occur in the submucous tissue of the œsophagus and the pericesophageal connective tissue, which, by suppurating and cicatrizing, lead to stricture.

Gummata of the gastric mucous membrane occur, but are often of slight clinical importance. In many cases, there is ulceration, but the ulcers are capable of cicatrizing.

The intestinal mucous membrane often has many gummata, which, if they ulcerate, leave cicatrized surfaces, and may produce perforation or peritonitis. Diarrhœa occurs in syphilitic persons, which is only arrested by specific remedies (syphilitic catarrh?).

Similar changes occur in the rectum; with the ulceration and cicatrization of gummata, and stenosis, symptoms of stricture are joined to those of chronic diarrhœa. The stools are usually composed of purulent, sometimes bloody masses; the finger detects cicatrices on the walls of the rectum. These things may easily be confounded with chronic dysentery, especially if tenesmus is present.

Besides gummata, broad condylomata have repeatedly been observed in the lower part of the rectum.

II. Treatment.—Local and general measures, based on the usual principles, are often assisted by surgery. Symptomatic treatment is often required, as passing a sound for stricture.

8. *Syphilis of the Liver.*

I. Anatomical Changes.—Severe disease of the liver is not rare; it almost always belongs to the later period of acquired syphilis, and is considered one of the so-called tertiary symptoms. But it is also found in children—as a rule, associated with hereditary syphilis.

The anatomical changes of hepatic syphilis are not always the same; they include syphilitic perihepatitis, hepatitis, and diffused interstitial hepatitis, gummous hepatitis, syphilitic cirrhosis, and amyloidosis.

For cirrhosis and amyloid liver, see Vol. II., pp. 201 and 213.

Syphilitic perihepatitis is marked by tendinous thickening of the serous coat of the liver and formation of adhesions of connective tissue to the neighboring organs—diaphragm, stomach, colon, wall of abdomen, etc. On section of the liver, it is seen that this often is connected with streaks of connective tissue in the interior of the parenchyma. It is rarely found alone; in such cases, it is distinguished from common perihepatitis only by the existence of lues in other regions, or by the excessive thickness of the affected tissue. It often causes the formation of depressions of the surface by cicatricial contraction of the new tissue, especially where it extends into the interior.

Diffuse interstitial syphilitic hepatitis, also called by E. Wagner diffuse syphiloma, is the commonest form of hereditary syphilis of the liver. In consequence of excessive interlobular proliferation of connective tissue, the proper parenchyma of the liver is destroyed and replaced to a variable extent by new connective tissue. The appearance of the changed parts has been compared to the color of flint (Gubler) or that of sole

leather (Trousseau). No lobular markings are seen—a condition usually but little observed in the liver of new-born children.

A whole lobe is sometimes changed to wheal-like connective tissue and wrinkled. Cicatricial retractions reaching to the surface of the liver are common in this case.

The microscope exhibits in syphilitic hepatitis a connective tissue very rich in cells, some spindle-shaped, others round. Careful examination will show that the portal branches are essentially involved in this process; their walls are thickened, and abound in round cells, the endothelium is proliferated in many places, with retraction or closure of the vessels. Similar changes occur in the intralobular capillaries, the gall-ducts, and (according to Rindfleisch, Hayem, and Baillard Lacombe) also in the lymph-vessels. The liver cells become fatty and granular, and perish.

In gummous hepatitis, we can distinguish two forms macroscopically, one miliary, and one in large tumors. The former easily passes into the latter.

The formation of gummous nodules is usually connected with interstitial proliferation of connective tissue, and thickening of the serous coat of the liver; the microscope shows every stage of transition.

In miliary gummous hepatitis, very fine yellow dots are scattered through the liver, in various numbers, and in size from a pin's head down to a scarcely visible point.

The microscope in this case shows clusters of round cells, which originate in the walls of the vessels, chiefly the portal vessels, but also in the gall-ducts, and probably the lymphatics. In some places, several small groups unite to form a larger one, showing how the miliary form may turn to the syphiloma with nodules. Giant cells have also been seen in the little nodules.

Hepatic gumma in large nodules forms a tumor from the size of a pea to that of an apple. It is most commonly seen near the suspensory ligament and the lower edge of the liver, which led Virchow to suspect that mechanical irritation might give rise to the selection of that spot. There is sometimes one tumor, sometimes there are from thirty to fifty, and more.

The gumma is seldom a separate, rounded body; its envelope of connective tissue usually sends out many ramified processes into the neighboring hepatic tissue. Fresh gummata have a gray-red surface of section; in older ones, caseation occurs in the centre, which turns dry, crumbly, and cheese-yellow. This material displays under the microscope a granular and fatty detritus, while the non-caseous periphery has accumulations of round cells. Softening, suppuration, and bursting of pus into gall-ducts occur exceptionally. Calcification may occur.

Where gummata project above the surface of the liver, and there are at the same time perihepatitic and interstitial proliferations and cicatricial retractions, the liver seems to be composed of many globular segments, whence the name of syphilitic lobed liver. Sometimes a protuberance is so constricted by the bands that it is only connected with the liver by a thin, movable pedicle.

Such of the parenchyma as remains undergoes the fatty and amyloid changes; the latter may extend also to the new syphilitic formations.

The formation of the large gummata agrees with that of the miliary variety.

Schüppel describes three cases as peripylephlebitis syphilitica, in

which the formation of syphiloma was very closely connected with the course of the larger portal branches.

II. Symptoms.—In many cases, morbid symptoms are entirely absent in syphilitic disease of the liver, and the anatomical changes are discovered accidentally at the autopsy. Even when symptoms exist, they are usually so ambiguous that we have no right to infer syphilis of the liver unless the history, or affections of the skin, bones, or mucous membranes, prove the existence of constitutional syphilis.

In syphilitic perihepatitis, peritonitic friction murmurs have been felt and heard; pain in the hepatic region is also mentioned; a restricted or suspended respiratory mobility of the organ is to be carefully looked for.

In the other forms of syphilitic liver disease, we have to consider pain, jaundice, and changes in the volume and superficial character of the liver. Sometimes there are symptoms of cirrhosis (splenic tumor, ascites), especially when large branches of the portal vein are narrowed or occluded.

Death may be caused by general marasmus; in excessive ascites, by suffocation; in rare cases, it is connected with symptoms of acute yellow atrophy of the liver.

III. Diagnosis.—Many syphilitic changes of the liver are unrecognized during life. In other cases, the nodular protuberances may lead to confusion with cirrhosis, adhesive pylephlebitis, abscess, echinococci, and carcinoma. The etiology is most important; if neglected, there is no certainty.

IV. Prognosis.—This is generally unfavorable. This is partly due to the fact that the patient does not usually come under treatment till he has reached a condition of profound marasmus, so that nothing more than symptomatic treatment is possible. Gummata of other parts are dispersed by iodide of potash and mercurials; we may presume the like in the present case. Irreparable injury may have been done already by cicatricial tissue closing gall-ducts and portal vein branches.

V. Treatment.—If the existence of this disease is probable, use iodide of potash, and mercurials—the latter always with care. The use of the baths and waters of Tölz or Oberheilbrunn, and brine baths, may be tried. If there is extreme marasmus, nothing but purely symptomatic treatment may remain.

Appendix.—Syphilitic disease of the pancreas is rare; sometimes diffuse connective-tissue induration, sometimes gummatous nodes; it is of no clinical importance.

9. *Syphilis of the Spleen.*

I. Anatomical Changes.—Splenic disease is very frequent in syphilis, and occurs in several forms.

Acute tumor of the spleen is an early symptom (see Vol. IV., p. 331).

It occurs, though not constantly, at the time of initial sclerosis, or during the appearance of the first general symptoms, and thus gives to the disease, in some sort, the stamp of an infectious disease. During the use of mercury it gradually disappears. As long as it remains, relapses are to be expected. There are no anatomical data, but the changes probably consist of hyperæmia and hyperplasia of the cells of the spleen, and

include the cases which Virchow called soft syphilitic tumor of the spleen.

Interstitial syphilitic lienitis has a chronic development and long duration, and is a late symptom. The organ is enlarged, hardened, and a section shows it to be crossed by uncommonly broad and abundantly developed bands of connective tissue. The capsule is thickened, and sometimes adherent to the neighboring parts. Similar changes may also be found at the same time in the liver, making it doubtful whether the splenic disease is a consequence of that of the liver, or a co-effect.

These conditions occur in congenital syphilis; great importance should be attached to that circumstance in making a diagnosis.

Gummous lienitis belongs to the third period of syphilis. As in other organs, nodes form, which may become as large as a walnut, but may be just the size of a pin's head. When fresh, they have a grayish color; afterward they become drier, yellowish, opaque, and crumbly. They are rarely encapsulated in connective tissue. They seem to be capable of partial absorption, and to disappear, leaving a callosity of connective tissue. Puckerings and deformities of the spleen sometimes occur.

Gold has observed with the microscope, endarteritis and endophlebitis obliterans, hyperplasia of the connective-tissue framework, and abundance of round cells scattered through the latter. Zenker found tablets of cholesterin; pigment crystals occur.

Amyloid spleen belongs rather to the marasmus which syphilis causes than to syphilis itself.

II. Symptoms and Treatment.—We can only recognize this disorder when a syphilitic person, with demonstrable gummata in other organs, has an enlarged and nodular spleen.

Treatment, iodine internally, iodide of iron, iodine baths.

10. *Syphilis of the Kidneys.*

Syphilis plays an important part in the etiology of disease of the kidney. We have encountered it in chronic nephritis of the parenchymatous and the interstitial sorts, and the amyloid kidney; even acute nephritis may be produced by syphilis. The diseases have no specific characters by which they can be recognized as syphilitic, but it often appears that the interstitial proliferation of connective tissue is very extended, causing deep cicatricial puckerings of the surface, which give the kidney almost an embryonal, *i. e.*, a lobular form. This is always suspicious, but we can hardly infer kidney-syphilis from it alone, unless there is syphilis of other organs. It is, of course, extremely important to know whether a nephritis is syphilitic or not; the prognosis will be more favorable if we can expect to control the disease with mercury and iodine.

Gummata are developed in a few cases; but they remain unrecognized during life. There may be the miliary or submiliary forms, easily mistaken for tubercle, or nodes as large as a pea, seldom as large as a bean. They are quite like those of other organs, often become caseous, are inclosed by growths of connective tissue, are usually multiple (in an observation by Key there were thirty in one kidney), may be most abun-

dant in the cortex or the medulla, and may cause great contraction, leaving little of the normal kidney remaining.

Treatment as for liver and spleen.

APPENDIX.—For the relation of syphilis to hæmoglobinuria and albuminuria, see Vol. II., p. 252.

11. *Syphilis of the Sexual Organs.*

The testes are often affected with syphilitic sarcocele. There may be thickening of the tunica albuginea with connective tissue, or interstitial proliferation of connective tissue in the septa between the seminal ducts or nodes of gumma in the substance of the testis. Cases of the latter sort may be confounded with cancer, but they are not painful, either spontaneously or on pressure, and cancerous degeneration in the neighboring lymph-glands of the groin is absent. We distinguish syphilitic sarcocele from tuberculosis by the fact that the latter always begins in the epididymis. Gummous nodes of the testis may soften and suppurate, the testis and scrotum adhere, and the pus is discharged externally. If both testes are affected, with inflammatory changes and adhesions in the vasa deferentia, impotence may result. But it is wonderful how small a remainder of a testis enables some men to be potent.

Gummata occur in the corpus cavernosum penis, causing sometimes loss of substance, cicatrization, and angular deformity.

The epididymis is rarely attacked by syphilis independently, but may be associated with the testis in the affection. So with the disease in the vas deferens, seminal vesicles, and prostate.

Treatment internal and local, according to rule.

12. *Syphilis of the Organs of Circulation.*

In the heart muscle, syphilis may cause the formation of circumscribed nodes, or diffuse infiltration, and callosities; these are often associated with disturbance of the power and functions of the heart, but are partially reparable (see Vol. I., p. 64).

In the endocardium, there may be thickenings which cause interference with the valvular action.

Arterio-sclerosis of the great arteries and aneurisms have been justly associated with syphilis; but the most important changes of this class belong to the middle-sized and smaller arteries, to be described in the following paragraphs.

13. *Syphilis of the Brain.*

I. ETIOLOGY.—This term includes not only affections of the parenchyma proper, but more especially those commoner ones of the meninges and arteries (some consider them the only ones), which involve the brain as it were secondarily.

Morbid symptoms of the brain or cerebral nerves are often connected with syphilis of the skull. For example, ulcerating gummata of the cranial bones may cause cerebral abscess; or syphilitic growths in the bony canals may compress the nerves and cause paralysis. Such occurrences are not mentioned in this chapter.

Intra-cranial syphilitic growths are very common indeed, more so

than in any viscus except the liver. In the majority of cases, perceptible changes belong to the late forms of syphilis, and must often be counted with the tertiary symptoms. Sometimes more than sixteen, twenty, even thirty years pass before the first symptoms of intracranial syphilis appear, and the patient is apt to express incredulity when told the truth. Exceptions, however, occur; cases are described in which brain-symptoms appeared within the first year after infection; Fournier says that facial palsy may appear directly after the hard chancre, but a knowledge of the anatomical changes is wanting.

Persons of an inherited nervous temperament are specially liable to brain-syphilis. Injuries to the skull often seem to have been the exciting cause. Excesses *in Baccho et Venere*, mental strain, and psychical excitement have been found to bear a certain relation to it. Lancereaux observed that the learned class furnishes a larger proportion of brain-syphilis than the laboring class. Slight or dilatory medical treatment of the primary disease seems to favor its appearance.

Broadbent says that the danger is especially great when the secondary symptoms were slight and the tertiary appeared very early.

Cerebral syphilis is of course most frequent in adults, but Graefe has demonstrated it in a child of two years. Virchow states that children may be born with signs of congenital encephalitis, arising under the influence of syphilitic infection of the parents and children; but Jastrowitz's investigations of the history of its development have thrown doubt upon this.

II. Anatomical Changes.—These chiefly affect the membranes and vessels of the brain. Many authorities consider the vessels as the chief sites, and as always forming the point of origin. It is doubtful whether syphilitic growths can spring from the cerebral parenchyma proper; they probably in all cases spread from the meninges or blood vessels into the brain.

In the meninges we find gummous tumors, often springing from the dura mater, and next from the subarachnoid tissue. Their origin at the dura is generally between the two layers, from which point they press outward towards the skull or inward towards the brain, or in both directions. They much prefer the top of the arch of the cranium and the base of the skull; in the former case, often lying on the falx cerebri; in the latter, on the prominences of the sphenoid bone, especially near and along the cavernous sinuses. The latter point is of importance as showing how certain nerves, most frequently the oculomotor, and next the abducens, are compressed and paralyzed.

Meningeal gummata may be circumscribed tumors like nodules or tubercles, or may shade in the most gradual way into the neighboring tissue, like inflammatory infiltration. In the former case, the size may be that of a hen's egg, though smaller sizes are the rule. Numerous miliary gummata have been found on the meninges in rare cases. The section presents a gelatinous consistency and a gray-red transparent look, or it may be dry and cheesy-yellow, or the centre may be cheesy and the periphery still succulent. There may be several cheesy spots in the central layers.

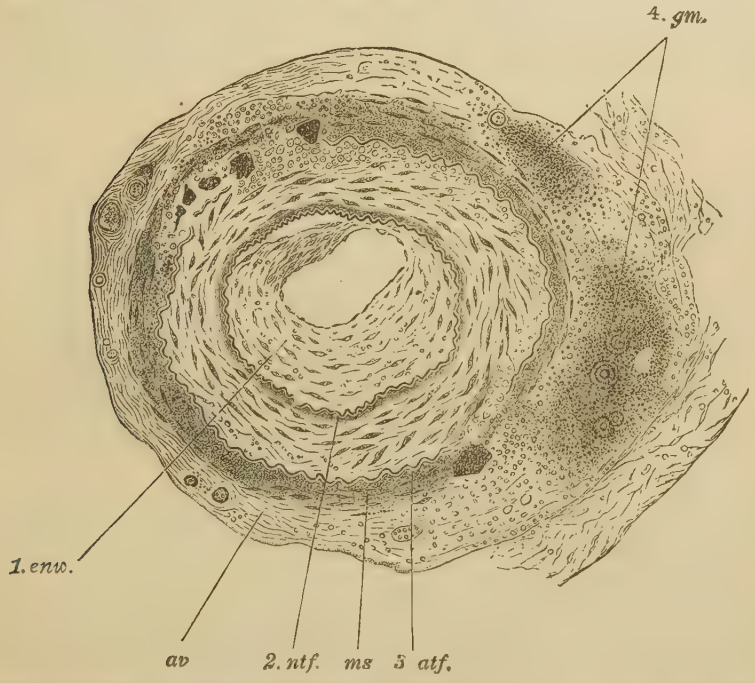
The effect on the neighborhood is that of pressure and consecutive atrophy; inflammation and softening may be added. The latter is especially the case with gummata that grow deep into the brain, with softening and destruction not confined to the new formation, but implicating the surrounding parenchyma to a greater or less extent. Very

considerable disturbance of the brain may result from this. The starting-point of such growths may not be discoverable. The meninges are often adherent, thickened, and everything so mixed together that it is impossible to draw distinctions.

When the gummous growth is an infiltration of the meninges, the latter are thickened, sometimes gelatinous and juicy, sometimes snow-white and of cartilaginous hardness.

Many believe that syphilis causes real inflammation of the meninges, not distinguishable from ordinary meningitis, at least not anatomically, though perhaps remarkable for a tendency to chronicity. Most recent authorities properly deny this. Much has also been said of the effect of hyperæmia of the meninges and brain in causing many disturbances, but this is little understood.

FIG. 66.



Syphilitic endarteritis of the arteria fossæ Sylvii, after Baumgarten. 1, *e n w*, endarteritic growth; 2, *n t f*, new-formed tunica fenestrata; 3, *a t f*, old tunica fenestrata; 4, *g m*, gummata in the adventitia; *a v*, adventitia; *m s*, muscular coat.

Petrow accounted for the disturbance of circulation in the meninges and brain by anatomical changes of the sympathetic nerve, but this will not stand before unprejudiced criticism.

Syphilis of the cerebral arteries involves two processes, gummous formations, and endarteritis, the former seated in the adventitia and media, the latter (corresponding with its name) in the intima. They have very close relations with each other, as Baumgarten has very well shown; the former may give rise to the latter. In other cases, the endarteritis may originate independently.

Syphilitic diseases of the vessels, to whichever form they belong, are

chiefly found on the great arteries of the circle of Willis. The basilar artery and that of the Sylvian fossa are especially attacked; hence symptoms of disease of the pons or implication of the third frontal convolution and island of Reil (aphasia) are strikingly frequent in cases of cerebral syphilis. The affection often embraces many vessels, and extends centrally towards the heart on the trunk of the common carotid, while peripherally it spreads to some of the vessels of the cortex.

The changes in question are discernible by the naked eye. The vessels are thickened, gray, gelatinous or tallow-yellow and opaque, round (not flattened like normal vessels), and presenting an open cavity when cut, instead of falling together. Transverse sections show the thickening of the vascular walls with especial distinctness. It may have gone to the extent of almost closing the vessel, or there may be a thrombus at the contracted part, which has caused necrotic softening of the part of the brain that is supplied by the vessel. The entire circumference is often involved; in other cases, the thickening is circumscribed, and may project inwardly like a button. It is evident how disturbance of func-

FIG. 67.

Syphilitic endarteritis of the basilar artery; 1, *enw*, endarteritic growth; 2, *tf*, tunica fenestrata; 3, *ms*, muscular coat; 4, *av*, adventitia. Enlarged 90 diam. Author's observation.

tion occurs, and also, how much more important a complete closure is than a partial one. The locality is important, for a blocked artery of the cortex may have its place supplied by anastomosing vessels, while one at the base of the brain leads to irreparable loss. Thickening may go to the extent of converting a vessel into a solid round cord, without the intervention of a thrombus.

Microscopical examination of the blood-vessels of the brain shows accumulation of round cells in the media and adventitia. Giant cells are found among them, as in gummata of other organs; afterwards the tumors may become cheesy.

Baumgarten's illustration (Fig. 66, 4, *gm*) shows that gummous processes may sometimes be united with those of endarteritis, though the latter may exist independently (Fig. 67). In endarteritis, a formation composed of branched cells, and sometimes containing round cells, pushes in between the endothelium and the tunica fenestrata in the tissue proper of the intima. On account of the strong tendency to proliferation and obliteration of the vessel, C. Friedländer has proposed the name endarteritis obliterans, but we prefer the term endarteritis proliferans. It is undecided whether the new cells originate exclusively from the vasa nutritia of the media and adventitia, that is, have emigrated (Baumgarten), or from a proliferation of the endothelium of the vessel (Heubner), but I have

repeatedly found the media and adventitia uninjured when the endarteritis was most pronounced. The process is sometimes terminated by the forming of a new endothelium on the inner surface of the growths and a new tunica fenestrata on the outside (Fig. 66, 2, *ntf* and 3, *atf*).

We must not suppose that endarteritic changes are characteristic of syphilis. They occur in other organs in a great variety of circumstances, as in the vicinity of inflammations and new formations. Syphilis is one of their many causes, and they are especially referable to syphilis when found in cerebral arteries. They are distinguished from arterio-sclerosis by their occurrence in youthful persons and by absence of fatty and calcareous changes.

Cerebral aneurism has been connected with syphilis. Aneurism of the aorta, or gummous and thrombotic changes of the heart muscle, may affect the brain by thrombi or portions of ulcerous surfaces becoming detached and carried to the brain as emboli.

Many authors state that encephalitis can develop directly as a consequence of syphilis; this is doubtful.

The idea that syphilis might cause purely functional disturbance of the brain originated at a period when the affection of the arteries was not known.

III. Symptoms.—They are marked by great variety. Either psychic, sensory, or motor disturbance may be prominent; or several groups of symptoms may be combined.

Psychical symptoms are common. The patient becomes irritable, whimsical, indifferent, loses interest in work, and may develop a pronounced mental disorder. Progressive paralysis of the insane is often connected with syphilis, though this is exaggerated by many, *e. g.*, Mendel. Syphilophobia is often present. The thought of being syphilitic is a constant torture; the victims spend their time going from one doctor to another to obtain assurance that they are free from disease; they neglect their family and business, and become misanthropic and hypochondriacal. This occurs in persons who never were infected, but are completely overpowered by the memory of an impure connection.

Loss of energy and weakness of memory are often associated.

Aphasia often appears very suddenly, lasts some hours (seldom days), and then sometimes disappears in a surprising way. Repeated attacks of aphasia without paralysis of extremities are characteristic of cerebral syphilis, and are to be associated with changes in the region of the arteria fossæ Sylvii and temporary disturbance of circulation (stenosis of cortical arteries, with speedy restoration of collateral circulation).

Many patients show marked somnolence, lying for hours or days in a half-sleep, like intoxication, either quiet or delirious. Such attacks may occur at very variable intervals. Awakening and clearing of consciousness usually take place very gradually.

Sleeplessness occurs in other patients, lasting for weeks and driving them almost to distraction; they cannot usually tell what prevents sleep, as they have neither pain nor disturbing thoughts.

Headache and neuralgiform attacks sometimes occur. The headache is diffuse or fixed in given spots, superficial or deep, increased or excited by knocking, or not influenced by so doing. Bodily and mental excitement, and excesses in Baccho at Venere usually render it intolerable. It may be so great as to cause furious delirium. It often occurs chiefly at night and destroys sleep. It sometimes disappears spontaneously, and so suddenly as to lead us to believe that it is due to states of fluxion. Neuralgia of the nerves of the brain or extremities may be both violent and obstinate.

Numbness, and many sorts of paræsthesia, are frequent; they occur in many different nerve-districts, sometimes distinctly circumscribed.

Paralysis of certain cerebral nerves deserves special attention. The motor oculi is most frequently affected; the abducens next. Sometimes both are paralyzed at once; perhaps one in one eye, the other in the other. Such paralyses may disappear spontaneously, or may be relieved very rapidly by iodide of potash (after the fruitless employment of electricity), but they often relapse. They usually owe their origin to gummous affections of the dura at the base of the skull. Some branches of the oculomotor often remain free.

The facial nerve may be the one attacked. All the branches are often attacked at once, indicating that the disease is peripheral; but in many cases there is rather a kind of weakness and laxness of the facial muscles.

The nerves of sense are sometimes affected. There may be loss of hearing, usually on one side only; or the sense of smell or sight suffers. The ophthalmoscope may show the latter to be purely functional (without visible changes of the optic papilla and retina), or associated with neuritis (J. Jakobson), neuro-retinitis, or atrophy of the optic papilla. In one case, endarteritis proliferans of the arteria centralis retinæ was demonstrated anatomically.

Liebreich and Förster report a rare form of syphilitic retinitis, comprising extravasation and formation of white spots in the retina.

Paralysis or conditions like it often occur in the extremities. They may be confined to one extremity (monoplegia), or to single groups of muscles; they may be unilateral or bilateral (paraplegia). They often occur with great suddenness while a certain motion is being attempted; or they may come on very gradually, beginning with fatigue and weakness. The condition of weakness and paresis is very often permanent. Frequent change in the distribution and extent of paralysis often occurs; going from one group of muscles, one extremity to another. The symptoms may last for hours, or days, or months.

Paralysis often appears in the extremities as an apoplectiform attack. The patient suddenly falls, the consciousness may be lost, or almost wholly retained; hemiplegia has occurred. The latter is often connected with aphasic symptoms, and right-sided; or symptoms of disease of the pons occur, since the region of the arteria fossæ Sylvii and the basilar artery are very often affected. It is very noticeable that the patient sometimes continues somnolent and almost in a dream for many days and weeks, with occasional conscious moments, followed by relapse. Excited states occur; tossing in bed, attempts to leave the bed and dress; there are false ideas, expressed in word and gesture. Death often follows after long somnolence or coma; or the patient recovers, but is carried off after one or several relapses, sometimes at short intervals. A surprising improvement in the paralytic symptoms often occurs, and sometimes complete convalescence; but if paralysis remains, there may be contracture (secondary degeneration) and atrophy from inactivity of the paralyzed muscles.

Among the motor disturbances, epilepsy is especially important. It may be exactly like the non-syphilitic disorder. It may differ in having no aura, in the very rapid succession of the attacks, in consciousness being only incompletely recovered during the intervals.

The contractions may be limited to one side or one extremity, may be unaccompanied by loss of consciousness, and may present the symptoms of so-called cortical epilepsy (see Vol. II., p. 194).

Many patients suffer from severe and obstinate tremor; others, from severe giddiness.

Chorea is often seen. I have seen it in a very severe form in a man and a woman, in both cases directly after the eruption of extensive roseola, and in both cases disappearing quickly after the use of mercury.

Cerebral syphilis is usually chronic, though an apoplectic attack may put a sudden end to life. Relapses, with different symptoms each time, and a great variety of combinations of symptoms, are the rule.

IV. Diagnosis.—It is usually easy to recognize cerebral syphilis when, on the genitals, skin, mucous membrane, bones or hairy scalp, we find cicatrices, spots of pigment, bone swellings, depressions, or loss of hair; or the disease is known to have occurred. One must be careful not to accept statements implicitly, for patients are apt to call soft chancre and even gonorrhœa by the name of syphilis, and on the other hand, they are very likely to lie about the matter.

In the case of married men, we must inquire whether their wives have had repeated abortions, whether they are childless, whether children have died early, or have had cutaneous eruptions, obstinate ozæna, scrofulosis, or rachitis, for all these things are often connected with syphilis on the part of the parents, especially the father.

In default of these signs, we rely on practical experience. One who has seen, examined, and rationally treated many cases soon acquires an instinctive perception of what is and what is not syphilitic. A description in words is not easy. Sudden paralysis of eye-muscles, transitory aphasia or palsy, protracted and frequently returning somnolence, a combination of very different groups of symptoms, none very fully marked, deserve careful notice. The nervous symptoms are sometimes so incompletely developed as to suggest hysteria—a suspicion which, as a rule, is justified only in women. In the case of epilepsy, consider whether there is a hereditary basis for that disease or other neuroses, whether it originated in childhood or adult age, whether injuries of the head, or cysticercus of the brain, can be taken into account. Originating in adult life, and accompanied by perversion of consciousness in the intervals, it is open to the suspicion of a syphilitic origin.

It should certainly be a rule to regard a doubtful case as syphilis and treat it as such; this will hardly ever do serious harm. The presence of disseminated or syphilitic choroiditis may sometimes enable a physician skilled in using the ophthalmoscope to infer cerebral syphilis.

V. Prognosis.—Though this is always serious, yet a rational treatment may often check the symptoms, or relieve them more or less completely. We are not masters of the situation; even in light cases, severe and fatal symptoms may suddenly appear. The prognosis depends on the symptoms, and is bad in proportion to the supposed amount of destruction and softening of the brain; it is worse in disease affecting the region of the basilar artery than in that of the arteria fossæ Sylvii.

VI. Treatment.—There is difference of opinion as to whether mercury or iodine is to be preferred. From much experience we prefer mercury first, and use iodine in a sort of after-treatment. When both are given at once, we have often seen extensive furunculosis.

We think a daily inunction with ʒ iv. of ungt. hydr. cinereum is the best method of administration. If coma or other severe symptoms

threaten, we increase the dose to ʒ iiss. or more. It is very important to continue the inunction for a long time after the disappearance of the symptoms, and to resume it from time to time as a prophylactic. A rag thickly spread with blue ointment may be worn permanently at that part of the skull below which we have reason to suspect a focus of disease.

After an energetic inunction course, let the patient take iodide of potash for weeks, or even months { ʒ iiss. : ℥ vi., one tablespoonful three times a day an hour after eating). Seguin recommends as much as 1¼ oz. of the iodide dissolved in water, one and a half hours before eating.

I have often seen brilliant success from the waters in Tölz and Adelheidsquelle-Oberheilbrunn, and Aix-la-Chapelle; sool-baths and indifferent warm springs (Wildbad-Gastein, Wildbad-Württemberg, Pfäffers, Ragaz, Teplitz, Schlangenbad) have also been praised.

As purely symptomatic treatment, electricity is used for paralysis.

14. *Syphilis of the Spinal Cord.*

I. Etiology.—There is no doubt that syphilis leads to diseases of the cord, but there is much difference of opinion as to the frequency of their occurrence. It is impossible to decide this question, for syphilis is so widely spread that many patients may at later periods of their life fall victims to purely accidental attacks of spinal trouble. The success of iodine or mercury is undecisive, for they are often valuable in non-syphilitic cases. And finally, the anatomical changes are rarely so characteristic as to form absolute evidence of syphilis. We believe syphilis of the cord to be frequent, but cannot attempt to give statistics.

The symptoms usually belong to a late period, occurring conjointly with tertiary symptoms or after the secondary ones have continued for a long time. Five, ten, fifteen years and more often elapse from the primary infection. The first symptoms of the secondary period are more rarely associated with spinal disease; the primary affection is said to have been connected with it. The anti-mercurialists say that those syphilitic patients who have been treated with mercury are most liable to it. Women are less liable than men, perhaps because the latter are exposed to other injuries which influence the outbreak of the disease.

II. Anatomical Changes.—Diseases of the cord may be caused by syphilis directly or indirectly, or may be functional in their nature. The indirect affections are secondary to disease of the bones or meninges—usually exostoses and carious processes of the vertebræ, or inflammation, thickening, or gummata of the meninges—which affect the cord by compression or by transference of the inflammation. The proper source of the changes sometimes lies farther off; for instance, syphilitic ulcers of the pharynx may extend back and cause disease of the vertebræ, meninges, and spinal cord. These changes are almost always chronic in their course.

Among the direct affections of the cord we have to name myelitis, tabes, sclerosis, and many would add atrophic paralysis and progressive muscular atrophy. There are a few cases of gumma of the cord. The syphilitic nature of these affections cannot be determined anatomically, though considerable implication of the membranes of the cord, with extensive thickening and adhesion, must be suspicious. The myelitis which sometimes occurs in the form of many small disseminated foci (Westphal)

are chiefly due to endarteritic thickening with more or less complete closure of the arteries, as was described under cerebral syphilis.

We sometimes have to do with purely functional disorder, as Weidner in particular has shown, under Gerhardt's guidance. In spite of severe functional disturbance during life, no change has been found in the cord or its surroundings at the autopsy. Acute ascending spinal paralysis has repeatedly been brought in connection with syphilis.

III. SYMPTOMS.—There is scarcely anything characteristic of syphilis. It was once thought that a predominance of motor and absence or slight development of sensory disturbances were characteristic, but this is certainly incorrect. There is sometimes compression-myelitis, sometimes various forms of myelitis or tabes, multiple sclerosis, progressive muscular atrophy, atrophic paralysis, unilateral lesion of the cord, or tumor of the cord. We must cherish a suspicion of syphilis if there is a history of it and if cicatrices are seen on the skin and mucous membranes; though we do not often go beyond a suspicion, as all these may be coincidences. A sort of instinct for diagnosis is acquired by practice. The signs of the spinal affection sometimes remain unobserved during life, because concealed behind symptoms of cerebral syphilis.

IV. PROGNOSIS.—More favorable than in non-syphilitic cases; but a permanent and complete cure is not very common. Relapses often happen if the treatment is broken off too early.

V. TREATMENT.—As in cerebral syphilis. Electricity with gymnastics may hasten a cure. At the beginning of mercurial inunction, the disease may seem to grow worse, but that should not discourage us from persevering.

15. *Syphilis of the Peripheral Nerves.*

Peripheral nerves are seldom the seat of gummata; they are more frequently compressed or infected by syphilitic formations of the meninges or bones, muscles or fasciæ. The symptoms are anæsthesia, paræsthesia, neuralgia, spasms, and paralysis, but it is astonishing what extensive changes may occur without functional disturbance.

A functional character, rather than anything anatomically demonstrable, seems to belong to the neuralgias sometimes observed at the onset of secondary symptoms or precursory of them.

16. *Hereditary Syphilis.*

I. ETIOLOGY.—Since acquired syphilis is no local disease of the sexual parts, but a constitutional infection, we need not wonder that syphilis is inherited. But the transmission goes no farther than to the first generation. A few cases exist which prove that hereditary syphilis in childhood does not protect from acquiring it in later years.

Syphilis is inherited from either parent. We cannot infer from which one it comes, from the organs affected in the child, as von Bärensprung once taught.

If the father or mother has primary or secondary symptoms at the time of procreation, the child will certainly have syphilis; if both parents have it, assurance is redoubled. If both have tertiary symptoms exclusively, the child usually is not born with syphilis, but is apt to be weak and atrophic, is liable to have scrofula, consumption, and tuberculous meningitis at a later period, and often dies early.

When the father has primary or secondary syphilis, the mother may remain well if no communication of syphilitic secretion takes place through lesions of the skin; yet she may give birth to a child affected with hereditary syphilis. But many think that the mother experiences a kind of infection shown by striking pallor, depression, and feebleness; and further, that she is not infected by secretion from her syphilitic child. The latter is not a fact; the nipple, if abraded, may receive the discharge of broad condylomata on the child's mouth and become syphilitic. It is also stated that a fœtus inheriting syphilis from the father may sometimes infect the mother *in utero;* this has been termed *choc en retour.*

Special consideration is due to cases in which both parents are quite well at the time of procreation, but the mother in some way becomes syphilitic during pregnancy. Many state that if the infection occurs during the first half of pregnancy, the child is infected through the mother's blood; but not if it occurs in the second half. Others think that the children are sometimes healthy, sometimes not, without much regard to the date of infection. It is certainly easy for the child to become infected during delivery by the mother's secretions, or subsequently, by suckling a breast covered with broad condylomata.

The whole subject of hereditary syphilis is by no means so plain as might be inferred from the above. Grünfeld, for example, reports a perfectly healthy child born of a father who was syphilitic at the time of procreation, no antisyphilitic treatment having been given the mother. I have seen the same in a syphilitic woman. Hutchinson reports that one of a pair of twins had signs of hereditary syphilis and died of it, while the other remained perfectly well. Families with the eldest children syphilitic and the younger ones not, are not an exception to rules, for the transmissibility grows less as time passes. Syphilitic and non-syphilitic children sometimes alternate; this is explained by the fact that the disease is usually transmitted only during the existence of active symptoms. Exceptions to this principle, however, occur. In short, there are the most curious freaks, and the greatest variety of forms, in the symptoms and the manner of communication of syphilis.

II. Symptoms.—Abortion or premature birth is frequent, the fœtus being born in a putrid and macerated state. The umbilical vessels, too, are often thickened, and the umbilical vein closed by thrombi. Thickening of the vascular wall affects chiefly the outer layers of the inner coat, and is parallel with endarteritis obliterans of the cerebral vessels (Vol. IV., p. 358). Interstitial proliferation of the connective tissue of the placenta occurs (interstitial placentitis, Oedmansson), with calcification and gummous nodes. Thus the death of the fœtus is caused by closure or contraction of the umbilical vein, which is the channel both for nutrition and respiration. Hereditary syphilis is the cause of the majority of abortions; whence comes the practical rule, that repeated abortion, especially of a putrid fœtus, excites suspicion of syphilis, and leads us to treat one of the parents specifically. The discerning physician can thus often win the lasting gratitude of unhappy parents.

In other cases, children are born alive, but die quickly with infantile athrepsia. They are wretched creatures when born, with pale, sunken faces, weak, bleating voice, sleepy, and weak in suckling. The skin is poor in fat, leathery, and wrinkled; the skin of the palms and soles thin and shining, as if glazed; there is often erythema of the nates. The autopsy shows syphilitic changes of the viscera and bones.

In a third class of cases, the children are born well, and at first make good

progress in development, but after a time display certain suspicious signs. They seem to have coryza. They snuffle, have difficulty in breathing through the nose, and often drop the nipple while sucking; they become blue; since new-born children cannot breathe through the mouth, they have a purulent discharge from the nose. Such things must appear most suspicious if they are of long continuation, in spite of care to avoid colds. Syphilitic disease of the mucous membrane of the nose is the cause.

Rhagades of the corners of the mouth, apparently spontaneous in origin, and very obstinate, are often nothing but broad condylomata in process of degeneration.

Syphilides very soon appear—roseola or papules. As in acquired syphilis, the latter often assume the character of broad condylomata in places where two surfaces are in contact; for this we look principally at the anus, groin, scrotum, navel, arm-pit, corner of the mouth, behind the ear, behind the ala nasi. Bullous and pustulous eruptions also occur. Bullous eruptions are known as pemphigus syphiliticus neonatorum; they are distinguished from the non-specific pemphigus (see Vol. III., p. 331) by the contents of the vesicles being more purulent and opaque, and the wall not tense, but often in loose folds and wrinkles. The palms and soles are the regular seat of the syphilitic form; if pemphigus is limited to those places, it may be assumed to be specific.

In the mouth and fauces, erythematous, roseolar, and condylomatous disease appears.

Many children suffer pain when the extremities are handled, perhaps owing to disease of the bones. They hold the extremities as quiet as possible. Iritis sometimes occurs, but is usually delayed for some months.

Hard chancre does not appear, so that we have a sort of syphilis *d'emblée*.

During these eruptions, the child usually loses strength; his healthy complexion, muscles, and fat disappear, the appetite is weak, vomiting and diarrhœa occur, and marasmus and death close the scene.

Children are sometimes, though not usually, born with these symptoms. They rarely appear within the first two weeks, but usually between the fourth and the eighth; they are rare after the third month; and, if the child has been completely well for the first six months, we may inform the parents that, in all human probability, there is no further danger. Late cases are believed in by some; children are supposed to have been attacked between the fourteenth and the sixteenth year for the first time; and women at the period of pregnancy! The fact is more than doubtful, for the cases described as syphilis hereditaria tarda seem sometimes not even to have been syphilitic, but rather tuberculous (scrofulous); and further, secondary symptoms may have been overlooked in infancy.

The disease may end with secondary symptoms in acquired as in hereditary syphilis. But tertiary symptoms may appear in early childhood —*e. g.*, in the liver or lungs; or they appear first at the time of the second dentition (seventh year of life), or during puberty. These include gummata, rupia, ulcers of the skin, disease of bone and joints, gummata with destruction of the hard palate, nose, etc. Laryngeal disease, as in acquired syphilis, may lead to great contraction of the larynx. Such changes are found even in the trachea and bronchi. It is not always easy to distinguish such disease from tubercle (scrofula).

The disease is sometimes concealed behind unusual symptoms, *e. g.*, ascites of doubtful origin, explained after death by affections of the liver and portal circulation.

Sometimes the sin of the parents is avenged by other diseases. Tertiary syphilis on the part of both parents at least predisposes children to such disease as tuberculosis, or chronic hydrocephalus, chorea, idiocy, epilepsy, etc.

III. Anatomical Changes.—These are of great weight in determining the nature of the disease, especially when the parents deny previous infection.

Of special value as signs are certain diseases of the epiphyseal ends of bones, and the ends of the costal cartilages, which appear with some regularity, and are sometimes the only sign of syphilis. The portion of epiphyseal cartilage lying close to the bone is much proliferated (zone of lime infiltration), and not bounded by a straight line, but forming notches and zigzags against the epiphysis. This may afterwards break down, so that epiphysis and diaphysis are separated, crepitation can be felt during life, and the child does not move the limb. In separation of epiphyses, cure is possible.

In serous cavities, bloody fluid is often found, especially in putrid fœtuses; there are also tendinous and cicatricial thickenings, wrinklings, deformities, and adhesions of the serous coats.

Great importance was attached by French authors, especially Dubois, to abscess in the thymus gland, which was supposed to be alone sufficient for diagnosis, but this has not proved correct. Virchow has expressed a reasonable doubt whether the juice of the thymus may not often have been taken for pus.

Gummata and interstitial and alveolar disease have been described in the lungs (see Vol. IV., p. 349). The lungs and liver are most frequently attacked by hereditary syphilis.

The spleen is often swollen.

The liver presents perihepatitic, interstitial, and gummous changes, just as in acquired syphilis (see Vol. IV., p. 351).

Birch-Hirschfeld has described interstitial proliferation of connective tissue in the pancreas. Förster described the like in Peyer's patches.

In the cortex of the suprarenal capsules, there are often found small multiple cheesy gummata. The kidneys also may contain nodules of gummata; also cysts.

The bones of the skull may exhibit inflammation and necrosis, and the meninges may be thickened. For congenital encephalitis, see Vol. III., p. 238.

IV. Diagnosis.—It is often easy to recognize hereditary syphilis. We suspect it if abortion, premature birth, and premature death by infantile marasmus occur repeatedly in a family. The post-mortem diagnosis must rest chiefly on alteration of bone.

We must examine a living child closely when we find obstinate snuffles, with purulent discharge, and rhagades in the corner of the mouth.

Hutchinson has pointed out two signs of previous hereditary syphilis visible in later years: a deformity of the two upper inner permanent incisor teeth, and disease of the cornea.

Deformity of the cutting teeth he refers to stomatitis, connected with syphilis, which makes the inner upper incisors lie, not parallel to each other, but converging or diverging. Their free edge is notched,

and gradually breaks off, forming a deep semilunar gap. These teeth are shorter than the others. The Germans place little faith in this indication.

Of Hutchinson's keratitis interstitialis diffusa (parenchymatosa, s. profunda), we can only say that it is very probably referable to past hereditary syphilis.

V. PROGNOSIS.—It is always serious. Many die of infantile marasmus, others have severe deformity or permanent impairment of special senses in tertiary attacks. Artificial feeding is almost sure to cause diarrhœa and loss of strength, which places the child's life in serious danger.

VI. TREATMENT.—A judicious prophylaxis enables us to bring more relief and happiness than is the case in almost any other disease.

Persons who have been infected with syphilis must not contract marriage for three years after; nor then unless they have been free from all relapses for six months past. They must, even then, always be on the watch for relapses, and avoid sexual intercourse at times when there are manifest signs of syphilis.

If several abortions occur in spite of this, a vigorous specific course of treatment must be given to one of the couple, usually the husband.

If a pregnant woman is infected in any way, an energetic treatment must be pursued during pregnancy. This holds good even if infection occurs in the second half of pregnancy, and a healthy child is expected—for infection is possible during parturition or subsequently. Inunction with mercurial ointment seems to us most suitable.

We have said that a healthy mother may have a syphilitic child. The question arises, whether such a mother ought to suckle the child. It has been stated that the child does not infect the mother in this case, but the statement is wholly false. We ought to say to such a mother that she has the choice between artificial feeding, which is sure death for the child, and the risk of personal infection in nursing. We must, of course, treat the child so as to relieve its symptoms as soon as possible; and if there are any abrasions or cracks of the nipples, we must order the child removed until they are perfectly healed. We may also give the mother iodide of potash (ʒ iiss. : ℥ vi., a tablespoonful three times a day), less as prophylaxis than as indirect treatment of the child through the mother's milk.

To tempt a poor woman with money to run the risk of infection in order to save the mother, is going beyond the line of a physician's duty. The person of one should be as sacred as that of another. If the nurse were already infected, and was unobjectionable in other respects, she might take the mother's place.

If both parents are well at the time of impregnation, and the mother is infected during pregnancy, she may have a healthy child. Such a mother must on no account suckle her child, lest it become affected, not by the milk, but by the secretion of broad condylomata on the nipple. The child must have the milk of a nurse or a cow. If the mother's syphilis is noticed during pregnancy, she must have a course of inunction with blue ointment, to avert the danger of infecting the child as it passes through the vagina and vulva.

For hereditary syphilis, give internally calomel (gr. $\frac{1}{6}$, three times a day), or hydrargyrum oxydulatum nigrum (Hahnemann's mercurius solubilis) in the same dose, and after every meal wash the mouth out with a clean soft linen rag dipped in a solution of chlorate of potash (1 : 40). Bathe the child daily in water at 28° R. (95° F.) in which ʒ ij. of sub-

limate is dissolved. A wooden tub must be used, for chemical changes may occur in a metal one. Let no part of the bath-water enter the eyes or mouth. Broad condylomata on the skin are to be sprinkled thinly with calomel, and covered with cotton.

For tertiary symptoms, iodide of potash or of iron is good; for external use, mercurial plaster for ulcerations; for ulcerations of the mucous membrane, pencilling with ℞ Iodi puri, gr. iss.; potassii iodidi, gr. xv.; glycerini puri, fl. ʒ ij. M.

PART III.

LEPROSY.

Elephantiasis Græcorum.

I. Etiology.—Leprosy can be traced back to the most ancient times in Egypt and India; it is supposed to be mentioned in the books of Moses. It is one of those diseases that have spread gradually from land to land; in the Middle Ages, there were so many lepers in Europe that they were collected and isolated in certain asylums called leproseries, leper-houses, lazar-houses. Nothing but the unsparing practice of this method has relieved Central Europe from this pest. Leprosy scarcely exists in our latitude; the cases are almost confined to persons who have lived long in the tropics and have brought the disease back with them. In Europe it exists in Norway and Sweden (where it is called spedalskhed), at some points in the Baltic provinces, in Hungary and Roumania, some parts of Spain, Portugal, and Italy, and in Greece and Turkey. It almost seems as if here and there germs of leprosy remained concealed. Vossius has reported two cases from the confines of Memel who had never left their own country. The disease usually keeps near the coast-line.

There is much dispute about the causes; but there is no better way to arrest its spread than by the strictest seclusion—a fact which seems to point to communicability. Formerly ascribed to climatic influence and the use of spoiled fish or grain, it has of late been discovered to be an infectious disease whose germs were first seen by Hansen, and then confirmed by others. They are small rods, called lepra bacilli, which in many respects much resemble bacilli of tubercle, and are regularly found in leprous foci (see Fig. 68). Experiments in artificial implantation upon animals have not produced general leprosy, but Neisser, Damsch, and Vossius succeeded in culture of the bacilli at the infected spot. Animals do not seem disposed to leprosy, and spontaneous outbreaks of the disease are unknown among them. The hereditary nature of leprosy is affirmed by many authors, but it is doubtful how this can be reconciled with the schizomycete theory.

Most cases occur between the twentieth and the fortieth year; rarely in childhood. Nothing is known of the manner of contagion. Many consider it safe to have intercourse with lepers, pointing to the fact that the attendants in leper-houses scarcely ever are affected.

II. Symptoms.—The disease runs a very insidious course. The duration of the incubation cannot be stated, for the prodromata appear so gradually that they are not at first recognized as such. There is often an intermittent fever, and the symptoms may then be mistaken for those of malaria.

The skin is most frequently affected with gradual development of infiltrations and nodular elevations—lepra cutanea (tuberosa s. tuberculosa). In other cases, individual nerve-trunks are first attacked with leprous infiltration and ganglionic swellings, producing sensory, trophic, and motor disturbances—lepra-nervorum (s. anæsthetica). Many cases are mixed forms; the tuberculous is often the basis, with the nervous supervening; the converse is rather less frequent. The mucous membrane of the throat, larynx, and trachea, the blood, lymphatic glands, testes, liver, spleen, and especially the conjunctiva and cornea, may be the seat of leprous disease.

The prodromal symptoms, resembling intermittent fever, often last a year or two, before the first signs of tuberculous leprosy appear. At first erythema appears, sometimes turning pale, again leaving brownish spots. The spots by degrees become hard and infiltrated, turning at last to large knobs and humps. The flexibility of the skin and the proportions of the body suffer from these processes. The face assumes an indifferent expression, the eyelids, nose, and ears seem thickened and deformed, and hang down, etc. A succession of nodes usually continues during many years. The patient may live long, and often dies of intercurrent disease. Leprous ulcers are often developed from the nodes, either spontaneously or in consequence of mechanical irritation. The surface of the ulcer is flaccid. Some nodes, meanwhile, may disappear spontaneously, and continual advance and retrogression may be observed. Febrile movement often precedes the outbreak of new nodes.

While these processes are going on, the mucous membranes of the nose, fauces, larynx, and air passages may develop similar ones. The nose becomes impervious, and stenosis of the larynx, from infiltration and tumors in the epiglottis, ary-epiglottidean folds, true and false vocal cords, may cause dangerous attacks of suffocation.

The voice may have lost its timbre long previously.

The lymph glands are involved; large swellings of the submaxillary, cervical, and inguinal glands are especially common.

Loss of hair, especially of the brows, occurs. Leprous nodes and infiltrations of the conjunctiva and cornea may occur, and finally cause loss of the eye.

Leprous disease in the liver and spleen has been hitherto found of no clinical importance.

In lepra nervorum, the diseased peripheral nerves may be felt as thickened, tender cords, knotted in places. Districts of the skin are at first comparatively hyperæsthetic, but afterwards become very anæsthetic, and not in respect to touch and pain only. Trophic disorders are often added. Bullæ, like pemphigus, called pemphigus leprosus, often appear; Gerhard and Müller have demonstrated large numbers of lepra-bacilli in their contents. Pigmental anomalies of the skin, white or brownish spots, are common. Want of secretion from the sweat and sebaceous glands gives the skin a dry and lustreless look. Finally, the muscles waste and contract. Marked palsy is rare. Single members, as fingers, toes, or entire extremities, sometimes drop off—lepra mutilans.

The disease often lasts many years. Death is caused by marasmus, suffocation, putrid infection by absorption of the pus from ulcerating nodes, or intercurrent disease.

III. Anatomical Changes.—The coarse changes consist of infiltration of the skin, mucous membrane, and certain viscera, with round cells, or the formation of knots of round cells. Many of these cells are very

large (so-called lepra-cells) and have vacuoles in their interior. They constantly contain leprosy bacilli. These are fine rods, one-third to one-half as long as red blood-corpuscles, and extremely like the bacilli of tubercle. They are rarely found free, but usually inclosed in cells, where they often cluster in thick balls (see Fig. 68).

Unna reports that the leprosy bacilli always lie free, and that the lepra cells are sections of lymphatic vessels.

In the fresh state they move actively. They are easily colored with aniline colors, but do not take Bismarck-brown (vesuvin). They can be easily demonstrated by the method of double staining which is given on p. 276 of Vol. IV. They are distinguished from tubercle bacilli by the fact that they liquefy coagulated blood-serum, which the others do not do, and that tuberculosis of the iris does not follow inoculation

FIG. 68.

Leprosy bacilli.

in the anterior chamber of the eye. They take aniline colors very quickly; tubercle bacilli do not.

Spores are often seen in them, one at each end; three or four spores in one; or a rod looking as if turned into spores. The bacilli have been demonstrated in the skin, mucous membranes, peripheral nerves, lymphatic glands, testis, liver, spleen, and eyes; also in the blood, usually inclosed in white blood-corpuscles.

Virchow has described the spleen as swollen and filled with countless white and gray granules, shown by the microscrope to contain great numbers of leprosy bacilli.

IV. DIAGNOSIS, PROGNOSIS.—The diagnosis is usually easy, especially in persons from the tropics. In the early stages, leprosy may easily be confounded with malaria.

Cures have been reported; but the prospect is hopeless.

V. TREATMENT.—Strict confinement of the sick certainly does most to restrict the disease. When it is developed, douches and inunctions

with mercurial, iodine, or ichthyol ointment are most to be recommended. It is said that creasote and salicylic acid have been known to do good when given inwardly. Patients ought, above all, to leave infected regions.

PART IV.

DIPHTHERIA.

Diphtheria is an infectious disease which occurs most frequently as a local affection of the throat, but may also attack other mucous membranes, as those of the larynx and nose. Attempts have been made to demonstrate the virus, but as yet not with decisive certainty; it may even be true that there are various forms of schizomycetes which cause diphtheria. From the analogy with the other infectious diseases, it seems certain that the cause must be of this nature.

We wish to state, however, that we are using the term diphtheria in its strictly etiological sense, a neglect of which has given rise to great confusion. For, in spite of the unity of causation, the coarse anatomical changes in different mucous membranes may differ greatly; on that of the throat, the exudation is for the most part deposited in the tissue itself, and cannot be removed from its surface, nor usually without taking away the mucous membrane itself. But in the larynx fibrinous exudations are common, which lie on the surface, and can be removed easily and without essential loss of substance. Virchow has termed the latter class of alterations croup, restricting diphtheria to the former. In the etiological sense, both conditions are the same.

It would be quite wrong to class every anatomical croup as a diphtheria in the etiological sense, for doubtless there are croupous inflammations without schizomycetes. The experimental proof of this statement is easily given by applying caustic ammonia, caustic potash, chromic acid, or substances having a similar action, or by causing the inhalation of hot steam, which produce a true croup of the larynx, in the anatomical sense. The result is the same in man. Palloni described a case of croupous inflammation of the laryngeal mucous membrane caused by inhalation of chlorine gas; and Reimer, a similar case caused by swallowing muriatic acid. Laryngeal croup has occurred after extensive burns. Croupous inflammation of the œsophagus has been caused by swallowing caustic ammonia. Kozlakoff and Stricker produced croup of the gastric mucous membrane in rabbits by introducing dilute ammonia into the stomach. Croupous changes of the gall-ducts are sometimes caused by the irritation of gall-stones; and croup of the pelvis of the kidney, ureter, and bladder may be caused by stone, or the use of irritating balsamics and diuretics. Croupous disease, therefore, has two forms —the diphtheritic and the non-diphtheritic—as regards causation. Primary bronchial croup is another case of the non-diphtheritic form.

Diphtheritic processes, in the anatomical sense, are not always diphtheritic as regards causation; this seems especially true as regards many affections accompanied by necrosis, developed during many infectious diseases, and often termed pseudo-diphtheritic. *Vice versa*, infection with diphtherial virus occurs which does not lead to diphtheritic or croupous changes of the mucous membrane in the anatomical sense, as will be explained under the next heading.

1. *Diphtheria of the Fauces.*

(*Angina maligna. Angina membranacea. Synanche s. Cynanche Contagiosa.*)

I. Etiology.—There can be no doubt that this disease is contagious for, apart from its frequent occurrence in epidemics, it is often observed that patients coming to a place previously healthy give rise to an epidemic; or that well persons acquire the disease when placed in contact with patients, as in nursing, in kissing, in using vessels or instruments that were not cleansed, by being coughed upon, etc., and even by being in the same room. Physicians have often been inoculated with the virus through accidental scratches. Successful experiments on animals also show the infectious nature of the disease. Trousseau made unsuccessful experiments on himself and two of his hearers; but such failures of contagion happen at times with diseases whose contagious properties are fully known.

Primary and secondary diphtheria of the fauces should be strictly separated: the former is an independent disease; the latter develops in the course of other infectious diseases.

The primary form occurs sporadically in large cities at almost all times, so that foci of infection do not become extinct. It may become endemic in limited circles, as asylums, schools, boarding-houses. In many towns, certain houses are known as diphtheria houses; a great many cases occur in them, or they form the starting-point of new epidemics. Such houses often have a bad site, imperfect disinfection of privies, stagnant water, close, unclean, crowded rooms, etc. Such conditions seem suited not perhaps to generate diphtheria originally, but to favor its multiplication, and perhaps to make the inmates more subject to the influence of it.

Epidemics are not dependent on the season of the year, climate, or weather. Cold weather, variable temperature, and changing winds scarcely favor the spread of the disease except by inclining the throat to inflammation, whereby the action of the poison is assisted. For the same reason, the disease is found in temperate and sub-tropical countries oftener than in the tropics.

The age has great influence on primary diphtheria. Children between the ages of two and seven are most commonly attacked, and the tendency diminishes every year thereafter. Nursing children seldom have it; mothers with diphtheria have even been allowed to continue to suckle their infants—though I have known of a mother infecting her new-born child.

Sex has little influence; in later childhood, boys are said to be oftener attacked.

The disease occurs oftener in the lower classes; yet victims have been repeatedly taken from princely houses within a few years past.

One attack does not protect from the second; a repetition is more the rule than the exception. Many have a permanently or temporarily increased predisposition, while others are strikingly exempt. Hypertrophy of the tonsils causes an increased tendency.

The mode of infection is often unknown. Diphtheritic products certainly often contain the virus; but since occupancy of the same room is sufficient to communicate the disease, we must assume that the patient's exhalations are contagious. Schools, boarding-houses, play-

grounds, etc., favor the spread of the disease. Lifeless objects, and probably also intermediate persons, may carry it. Food from infected places, *e. g.*, milk (Klebs), has often spread the disease. It occurs in animals (hens), and Lutz and Limmer report its transference to men; Gerhardt and Seeber state the same.

It is a much debated question whether the infection begins in the fauces, in a purely primary form, becoming generalized afterwards, or whether the reverse is the case. An unprejudiced clinical study shows that both methods occur, for severe general symptoms often precede the local disease of the tonsils by days, while in other cases—in my experience more rarely—the reverse occurs. It is not impossible that these two courses represent different poisons. A primary generalized affection may represent infection through the respiratory tract or the gastro-intestinal tract, by eating infected food or by inhaling the air of a sick-room. The frequency with which the tonsils are attacked may be connected with the fact discovered by Stöhn, that there are spaces in the epithelial coat through which a continual passage of amœboid cells normally takes place.

Of the nature of the diphtheritic poison nothing certain is known. It is suspected to consist of schizomycetes, which are certainly found in the products; but among the many forms of fungi it is very hard to find the right ones. Klebs attempted to distinguish two varieties of throat diphtheria botanically, which he called the microsporine and the bacillary forms. They differ also clinically. Löffler, a profound student of the subject, is inclined to see the fungus in certain bacilli which are about as long as tubercle bacilli, but twice as thick. They are often composed of several members, bent or straight. Slight knotty thicke ings are often seen at the points of contact. The ends of the rods are stained more deeply with alkaline solution of methylene-blue than the other parts. Löffler cultivated this fungus purely, and successfully transplanted it to animals.

Secondary diphtheria of the throat is a consequence of infectious diseases. It is most frequent in scarlatina, but occurs in measles, German measles, whooping-cough, typhoid fever, small-pox, and erysipelas. Löffler has shown that in scarlatinal diphtheria of the throat the same organisms are found as in primary diphtheria, but it is not certain that this is always so. The nature of secondary diphtheria of the fauces needs further study.

In what follows, only the primary affection is discussed.

II. Symptoms.—The incubation period usually lasts from two to seven days. A longer period than this is commoner than a shorter one; many extend it three or four weeks.

The first symptoms may be general or local, or both. In either case they may begin violently or gradually and insidiously.

In children, a very sudden high fever (over 40° C.) is not uncommon, with all its preliminaries and concomitants: cold, livid skin, shivering or rigor, vomiting, convulsions, delirium, loss of appetite, thirst, etc.; on the next day, the fever is less or has gone, the diagnosis of ephemera is made, and ascribed to errors of diet, but diphtheritic changes of the fauces appear at the same time or soon after. It is therefore a duty to examine the throat with great care whenever such symptoms occur.

As a warning example of the treacherous course of the disease, I give an account of a case that I lately saw in consultation. A strong boy of four years, ill for a week. No special complaint except slight shivering. Appetite entirely gone. Pallor and loss of strength increasing. Sleepiness. For twenty-four hours, very feeble heart-sounds, with irregular

rhythm; striking want of fulness of pulse. Cause unknown. At the consultation, all the throat and part of the tongue and cheeks were seen covered with a thick diphtheritic layer. Death occurred in ten hours with symptoms of paralysis of the heart. Thus, in throat-diphtheria, local troubles may be absent; and it is a gross error to omit examination of the throat when there is no pain or difficulty in swallowing.

Among local subjective symptoms, the commonest is trouble in swallowing, usually accompanied by pain in speaking. Others have less pain in swallowing than in moving the lower jaw. The pain is located by the patient, not back in the throat, but lower and close behind the angles of one or both jaws. It often reaches to the region of the ear. Swollen lymphatic glands are sometimes felt behind the angle of the lower jaw which are sources of pain. I have treated patients in whom a change in the voice to a nasal tone was the only thing noticed, and was the reason for calling the physician.

Symptoms of laryngeal croup, originating in latent throat-diphtheria, sometimes take the first place.

Among the objective symptoms, those of the throat are the chief. It is possible that in severe constitutional infection a patient may die before the throat-lesions are developed; in such an event it would not be easy to make the diagnosis.

The affection of the throat is not always the same. The lightest form is catarrh of the fauces, which differs little from the ordinary sort, and may be diffuse, or limited to spots. The specific nature of this catarrh is evidenced by the existence of previous exposure to contagion and the power of communicating diphtheritic diseases to others. In the catarrh, swelling may be a prominent symptom; or redness; the secretion is not usually prominent. In circumscribed catarrh, the pain is usually limited chiefly to the diseased place. I have noticed that some persons seem to have a sensitive spot in their throats, to which, in subsequent attacks, the catarrh is apt to return. The catarrh is often only the precursor of further changes; in other cases, it disappears.

A second sort of throat-diphtheria consists of circumscribed superficial diphtheritic deposits. They are yellowish or grayish spots, very often confined to the tonsils, but also affecting the palate, posterior wall of the pharynx, and uvula. I have repeatedly seen them appear first on the tip of the uvula. They often originate in the follicles. They can usually be wiped off easily with a brush or the finger wrapped in a cloth, leaving slight loss of substance, if any; the base of the excavation being often tallowy-gray, and the edge usually sharp and very red. The deposit, under the microscope, consists chiefly of epithelial cells, schizomycetes, and a few round cells. The coating may re-form in a surprisingly short time. In a certain case, where I most carefully cleansed the spots every hour by pencilling with carbol-glycerin, I often found the deposit after twenty minutes as heavy as before.

Diffuse deep throat-diphtheria usually proceeds from the preceding form. The spots grow in size and become confluent; at the same time they become thicker and are harder to remove. They may extend so deeply in the tissue as to pierce the soft palate, uvula, or hard palate; or the uvula or tonsils may become separated, perhaps causing very dangerous hemorrhage. Deep scars may be left after recovery. The microscope exhibits an abundance of round cells in the diphtheritic mass, in addition to the elements above named.

The parts affected sometimes become a brownish, fetid, soft, and

pulpy mass. The stench is such that the diagnosis of septic diphtheria can almost be made with the nose. Extensive gangrene of the parts around may originate in the diphtheritic parts.

Swelling of the submaxillary lymphatic glands is usually observed in all forms of diphtheria; those behind the angle of the lower jaw are most constantly swollen and painful, forming a sympathetic bubo, *i. e.*, one due to absorption of products of diphtheritic inflammation by the lymphatic vessels and the nearest glands. They prevent the motion of the jaw, make movements of the head difficult, and if confined to one side may cause a sort of wry-neck. Absorption is usually complete after the diphtheritic process is past; suppuration is exceptional.

In many cases, all the submaxillary lymphatic glands take part in the inflammatory swelling; the cervical cellular tissue may also take part, forming beneath the jaw a diffuse swelling, as hard as a board, and usually of an alabaster color. This condition is dangerous in more than one way. It interferes with the opening of the mouth, and is an impediment to treatment. Compression of the air passages may cause danger of suffocation. There may be tedious suppuration and gangrene, erosion of large vessels and death from hemorrhage, fistulous formation leading into the mediastinum and serous cavities, etc. Finally, rapid death from œdema of the glottis may occur.

Diphtheritic changes have often extended to neighboring mucous membranes. The nose is affected rather frequently. The nostrils become stopped up, so that the patient has to breathe through the mouth; the false membrane is sometimes blown out, and may even be seen from outside; but a specially characteristic feature is the discharge of a putrid, almost stinking, light brown-red ichor from the nose, excoriating the upper lip and causing erythema and œdema.

The diphtheritic change sometimes attacks the mucous membrane of the nasal duct and the conjunctiva: in the former case producing epiphora, and in the latter being obvious to view. This complication is very serious, and may cause loss of sight.

The Eustachian tube, tympanic cavity, and even the external meatus are often attacked; the symptoms are deafness, subjective noises, severe pain in the ear, and often increase of the bodily temperature. This may cause loss of hearing, extensive suppuration of bone, thrombosis of sinuses, meningitis, and brain-abscess.

Extension to the respiratory passages usually causes symptoms of laryngeal croup.

The œsophagus and the mucous membrane of the stomach and intestine are seldom diphtheritic; the mucous membrane of the genitals is sometimes affected, perhaps by auto-infection.

Wounds easily assume a diphtheritic coating; this often occurs in children when the mouth has been forcibly opened, causing injury to the lips, gums, tongue, or hard palate. Rhagades of the lips are sometimes covered with false membrane. Slight injuries, as leech-bites or blisters, may undergo the same change.

During epidemics, diphtheria of wounds may appear in persons otherwise unaffected; the throat-disease may appear subsequently.

The general symptoms do not always agree with the local. We must especially avoid the error of supposing that cases with slight visible local disease are always light.

The bodily temperature hardly ever fails to be elevated, though not always to the same degree; in some, rising far above 40°; in others, but little above 38° C. There is no special type in its course. Low temperatures are not by any means a favorable sign; the septic forms often have low temperatures, though there is severe general infection, due to absorption of putrid diphtheritic masses, with almost always fatal results. The temperature of collapse is not rare, and is, of course, unfavorable.

The pulse often corresponds with the temperature. Intermission, or unusual retardation (as low as 50) may occur, and is always a bad sign.

There is quite often a moderate swelling of the liver and spleen, with sensitiveness to touch.

Advanced leucocytosis is sometimes found in the blood. Micrococci have been repeatedly found in the blood, first by Hueter and Tommasi-Crudeli.

The course is usually acute; the matter is usually decided by the enn of a week, or at the furthest, two. Some cases are subacute; I have seen two that lasted eight and ten weeks respectively.

Complications are not rare. Those due to transference of diphtheria to other mucous membranes have been mentioned. Others may be considered in part as metastases—the poison being carried to other organs by the blood and animal fluids.

Painful swelling of the salivary glands may occur, and salivation remains as a sequela.

In many cases, there is repeated vomiting, either at the beginning or continued through the disease. Diarrhœa sometimes occurs, hard to control, and increasing the danger of collapse. Peritonitis has occurred.

Albuminuria is not unusual, often appearing during the first few days; its significance varies. If slight, transitory, and limited to the period of fever, it is simply febrile albuminuria. If independent of fever and marked, and associated with tube-casts and round cells, epithelium from the urinary canals, and red corpuscles, it indicates nephritis. The amount of blood in the urine is sometimes so great that its presence is shown by the color. Hæmoglobinuria and hæmatinuria (Salkowski) also occur. Acute nephritis may be accompanied by a greater or less amount of anasarca, but this is not common—much less common than in scarlatinal nephritis. Anuria is sometimes associated, but uræmia rarely.

Cloudy opacities have been repeatedly found in the urine, composed of schizomycetes—the same have been described as forming a layer on kidney-casts. These things should be understood, as injurious seminal losses are a possibility.

There are sometimes very threatening heart complications. The sounds of the heart are soft, weak, the first sound seems muffled, the apex-impulse diminishes in force, the area of dulness rapidly enlarges, especially to the right, the action of the heart becomes irregular, and often very slow, and at last death occurs from paralysis of the organ.

Endocarditis, pericarditis, and pleurisy are rarer.

Death by asphyxia may occur if the muscles of respiration become palsied and expectoration is insufficient. Central disturbance of innervation may make the breathing irregular and sighing.

In many cases, sudden attacks of fainting occur, sometimes causing

death directly. They are apt to take place when the patient rises quickly, or sits up to perform the offices of nature, while in a weak condition. The cause is doubtless weakness of the heart and consequent anæmia of the brain.

Dangerous hemorrhage may occur, *e. g.*, from the nose, or when sloughing of the throat has opened blood-vessels. There may be symptoms of dissolution of the blood—hemorrhage under the skin or in many of the mucous membranes. In complicating endocarditis, cutaneous hemorrhages may be caused by embolism.

There are certain changes of the skin of less importance. In many cases, extensive erythema is one of the first symptoms, reminding us of scarlatina. There may be exanthemata like urticaria or (more rarely) papulo-pustular forms. Gangrene of the skin is much more serious. Erysipelas of the face has been seen. Erythema nodosum occurs as a sequela. Calimani states that he has found diphtheritic alterations of the bed of the nail, which he considers an absolutely unfavorable symptom.

Swelling and pain of the joints are rare; in my own cases, the knee has been affected, but the small joints of the fingers and toes have also been attacked not infrequently. Acute articular rheumatism may be fully presented. In a case of mine, acute endocarditis with mitral insufficiency followed it, and during a second attack of diphtheria a year later, polyarthritis occurred again.

Complications and sequelæ are not always easily separated, and may pass imperceptibly into each other. Anæmia, with obstinate loss of appetite, often remains, and in spite of all care, continued it may be for weeks, death from exhaustion is unavoidable.

Acute nephritis may either form a regular sequela, or the inflammation accompanying diphtheria may outlast the latter disease.

Especial attention is due to post-diphtheritic paralysis, affecting single viscera, or the muscles of the trunk or extremities. It usually appears in the second or third week after the disease, or even later; it specially affects adults, children between two and six being more rarely attacked.

Paralysis of the palate is common, known by the nasal voice, immobility of the hard and soft palate during swallowing and speaking, and by things swallowed coming out at the nose.

The eye often suffers, especially as to the apparatus of accommodation. The patient loses the power of reading or seeing clearly close at hand, and according to Jakobson's observations, hypermetropia increases. Both sides are almost always affected, though one eye is often worse than the other. Paralysis may appear quite suddenly, or may be observed accidentally at school in reading or writing. It is often combined with paralysis of other parts, and lasts weeks or months. Gradual improvement, and at last recovery, almost always follow.

Donders remarks that the pupil is often remarkably dilated, and reacts well to the stimulus of light, but slowly to changes of accommodation. Bilateral paralysis is probably of a peripheral origin.

Single muscles of the eye are sometimes paralyzed, and the affection often skips from one to the other muscle or eye. Bilateral palsy is rare (Uhthoff, Mendel).

Neuro-retinitis and atrophy of the optic nerve are said by Bouchut to

occur under the influence of diphtheria. Amblyopia and transitory amaurosis are mentioned, probably connected with nephritis.

Paralysis of the œsophagus sometimes occurs, causing great difficulty in swallowing; the patient has to be fed through a sound.

Paralysis of the superior and inferior laryngeal nerves is more frequent. In the case of the latter, paralysis may be restricted to distinct groups of muscles—as the posterior thyro-arytenoids or the aryepiglottici. Paralysis of the latter is easily recognized by failure of the epiglottis to cover the larynx in swallowing, so that food enters the larynx. If the mucous membrane of the larynx has lost its sensitiveness by paralysis of the inferior laryngeal nerve, coughing does not occur, and the foreign substances may enter the lungs, causing a variety of pneumonia, with gangrene or abscess of the lungs.

Sometimes only those fibres of the vagus are paralyzed which preside over innervation of the bronchial muscles or the heart. In the former case, the secretions may accumulate and suffocate a patient; in the latter, paralysis of the heart may cause death. Heart-failure may proceed from changes in the heart muscle also.

Paralysis of the phrenic nerve, if double, causes certain death by suffocation.

Paralysis of the vesical and rectal sphincters may occur. Sexual impotence (virile) has been described (Guillemant).

Paralysis of the extremities sometimes affects single muscles, sometimes entire limbs. The facial nerve may be paralyzed. Paralysis often ascends from the legs to the face, and may then be accompanied by severe anæsthesia. Cerebral hemiplegia, sometimes associated with aphasia, is rare.

The electric test for excitability often proves that the paralysis is of peripheral origin, but central disease may also be the cause.

These paralyses almost always recover, though months may elapse. Residual paralysis is not common, and is usually associated with atrophy.

Loss of the patellar reflex sometimes occurs long after recovery, and lasts a half a year or more. It may be confined to one side. Acute ataxia is not rarely developed in addition, connected with anæsthesia of the lower extremities and tottering when the eyes are shut, and may copy acute tabes dorsalis so closely that it might be mistaken for it in adults if the history were unknown. E. Remak properly states that reflex rigidity of the pupil (see Vol. III., p. 109) is absent. It is not known whether this is due to changes in the posterior columns of the cord.

Mania (Minot), epilepsy, chorea, and valvular lesion are rare sequelæ. I do not know from personal observation whether kidney diseases become permanent.

III. Anatomical Changes.—Numerous hemorrhages are very often found in the serous membranes, and in a great many other parts.

The muscles are sometimes very pulpy in consistency and dull yellow, and the microscope shows granular cloudiness and advanced fatty change.

Extensive changes are sometimes seen in the lymphatic glands: swelling, hyperæmia, and bleeding. Micrococci have repeatedly been found in the lymph-spaces, and Bizzozero has found small necrotic foci.

The blood is sometimes of a brownish color, thin and fluid. Increase of the number of colorless corpuscles and micrococci have been observed.

The heart cavities are sometimes much dilated, and the tissue lax. The right auricle especially may contain thrombi adherent to the walls, which may cause embolism. In spots, the heart-muscle is dull-yellow, from fatty change of the fibres. With the microscope, we often find extensive changes: groups of micrococci, fissuring of muscular fibres, Zenker's degeneration (Rosenbach), proliferation of nuclei in the muscular fibres, and disappearance of muscular substance with deposit of pigment (Leyden). On the endocardium, fresh inflammatory changes are sometimes seen. Klebs found interstitial changes in the lungs.

The changes in the spleen are like those in the lymphatic glands. The liver has been found fatty. The gastro-intestinal tract often has swelling of the lymph follicles, sometimes forming small ulcers.

The kidneys vary much according to the period, the severity of the disease, and the character of the epidemic. They may be of normal size, or swollen and enlarged. They are sometimes thickly dotted with hemorrhages; at other times they are pale and gray-yellow. In the lightest cases, the changes seemed confined to granular cloudiness of the epithelium cells of the convoluted tubes. If the disease progresses, interstitial changes are added. In the urinary canals of the medullary substance free desquamation of the epithelium, and in places dilatation of the canals occur. Swelling and multiplication of the epithelial cells and nuclei of the vascular loops in the Malpighian capsules also occur. Extensive fatty degeneration with increasing interstitial nuclear proliferation finally appears.

Schizomycetes have been repeatedly described in the blood-vessels, Malpighian capsules, and urinary canals, and the connection between throat-diphtheria and nephritis has been supposed to be that the kidney, in the act of excretion of the parasites, suffered irritation of its tissues. Fürbringer does not confirm this.

In the brain and cord, meningeal and parenchymatous bleeding is not rare. The microscope shows more. Klebs found the adventitious lymph-sheaths of the vessels full of schizomycetes, while in the cord Déjérine observed an accumulation of colorless corpuscles in the same places. This author states that in the spinal cord swelling of the ganglion cells of the anterior cornua, disappearance of the processes, and finally destruction of the ganglionic cells, and nuclear proliferation in the neuroglia of the gray substance take place, and are the cause of paralysis.

In the majority of cases, the palsy is probably due to changes in the peripheral nerves, which may be quite extensive, as in one of Meyer's cases. Parenchymatous degeneration and atrophic appearances are sometimes associated with multiplication of nuclei, formation of fat-granule cells, and vascular dilatation in the interstitial tissue. P. Meyer also described nodose thickening of the nerves—neuritis nodosa. Leyden showed multiplication of nuclei in the paretic muscles of the palate, with atrophic wasting.

The diphtheritic deposits consist in advanced cases of a fibrinous exudation, inclosing remains of epithelial cells and schizomycetes of many shapes and sorts, extending to a varying depth in the tissue beneath the epithelium, and set off from the sound tissue by a layer of colorless blood-corpuscles. There is a good deal of extravasation. Löffler's diphtheria bacilli are found arranged in little groups somewhat below the surface of the membrane, surrounded by numerous cells; they never

work into the tissue of the mucous membrane, and have not been demonstrated in other organs.

IV. Diagnosis.—The presence of the membrane leaves no doubt; but the catarrhal form can be interpreted as diphtheria only during epidemics.

Nephritis or specific paralysis sometimes enables us to infer previous diphtheria. Membranes caused by irritants are known by the history. Angina herpetica (Vol. IV., p. 134) is not usually hard to distinguish.

V. Prognosis.—We must in all cases be very cautious; in spite of slight local disease, the most dangerous complications may suddenly arise. Local and general symptoms do not always correspond; yet extensive local disease, and especially implication of the wall of the pharynx, implies severe cases. Septic and gangrenous cases are almost absolutely unfavorable. The younger a person and the weaker the constitution the worse is the situation. Every complication makes the prognosis worse, especially croup or laryngeal diphtheria. The connection with extensive nasal diphtheria seems equally fatal. Abundance of albumin in the urine is unfavorable. The prognosis is essentially dependent on the character of the epidemics, some of which give fifty per cent of deaths. Sporadic cases are usually lighter than epidemic cases.

VI. Treatment.—The strictest isolation of patients from well persons must be carried out. In epidemics, all persons with apparently simple catarrhal angina must be excluded from school and from general intercourse, and must be considered diphtheria patients. After recovery, no one may resume intercourse with the world before the clothes and bed-clothes have been disinfected; the sick-room must be disinfected also.

Family physicians should have patients teach their children when quite young to let their throats be sprayed, and to gargle. I know a number of cases in which children were the first to discover their own disease, and know of others in which medical treatment was rendered almost impossible by the refractory conduct of the children. Permanent enlargement of the tonsils ought to be relieved by excision as early as possible.

Physicians must look out for themselves, especially if they have cuts on the hands. If diphtheritic material has been injected into the eye, they must at once wash it thoroughly with disinfectants.

The removal of patients to wholesome and unsuspicious quarters often has astonishingly good and speedy results, but circumstances do not often allow it.

The food should be exclusively liquid, and nourishing: lukewarm milk, egg, meat-soup, beer, and abundance of wine, which is the most trustworthy agent for relieving the general symptoms. Spray the throat carefully once an hour with salicylate of soda (ʒ ss.: ʒ vi.); if the nose is affected, irrigate it from in front, at the same interval. Keep the patient in bed, let him always use a bed-pan, and avoid quick movements on sitting-up. Otherwise, purely symptomatic treatment.

There are a great many treatments and views about treatment. We mention the following. *a.* Disinfectants—carbolic acid, salicylic acid, salicylate of soda, benzoate of soda, permanganate of potash, creasote, iodoform, sulphur, sublimate, arsenic, etc., for pencilling, insufflation, gargling, parenchymatous injection into the tonsils, or internally. *b.* Caustics—sulphate of copper, muriatic acid, chromic acid, chloride of zinc, etc. *c.* Astringents—acetate of lead, subnitrate of bismuth, nitrate of silver, tannin, alum, etc. *d.* Balsams—turpentine,

cubebs, copaiba, eucalyptus, etc. *e.* Antiphlogistics—bits of ice to swallow, ice-cravat, inunction of mercurials, calomel, leeches. *f.* Bromine and iodine preparations—their vapors for inhalation, iodide of potash internally, tincture of iodine for pencilling. *g.* Solvents of the membrane—lime-water, lactic acid, neurin, pepsin, trypsin, papayotin, tetramethylammonium-hydroxyl, tetramethylen-ammonium-hydroxyl, etc. *h.* Chlorate of potash internally and as a gargle, quinoline, pilocarpine, peroxide of hydrogen, inhalation of caustic ammonia. *i.* Emetics.

2. *Laryngeal Diphtheria.*

(*Diphtheritic Croup, Angina Membrancea, A. Laryngea, A. Polyposa, Cynanche Stridula, Laryngitis Fibrinosa, L. Croupsa.*)

I. Etiology.—Croup includes every fibrinous inflammation of the laryngeal mucous membrane, which leads to the formation of superficial fibrinous membranes, usually removable without loss of substance. Laryngeal croup is not always diphtheritic in origin, *i. e.*, caused by the diphtherial fungus; it may be produced by chemical or thermic irritation. The diphtheritic and the non-diphtheritic agree closely in their symptoms, which point to increasing stenosis of the larynx. The following description relates to the diphtheritic form alone.

The two sorts, primary and secondary, must be distinguished, but the latter is of the chief importance, and many authors even deny the existence of the other.

Secondary laryngeal diphtheria is most frequently a sequel of throat diphtheria. The affection of the throat may be very insignificant, sometimes involving only the posterior surface of the uvula or the higher part of the posterior wall of the pharynx, where it is not seen, and hence the disease of the larynx is considered as primary. The younger a child is that is attacked by throat diphtheria the greater the danger of laryngeal diphtheria as a secondary affection.

Trendelenburg and Oertel have proved that diphtheritic poison may flourish in the mucous membrane of the larynx; Löffler found diphtheria-bacilli in the deposits. In man, the affection is chiefly due to transference of inflammation from the throat, but accident may come in play, as aspiration of diphtheritic membrane, or the flowing down of secretion into the larynx.

The disease sometimes accompanies other diseases, chiefly measles; but it may also follow scarlatina, whooping-cough, pneumonia, typhoid fever, relapsing fever, varicella, variola, cholera, and pyæmia.

The rarer, primary form, is more commonly sporadic than endemic or epidemic. The epidemic occurrence of primary diphtheria of the larynx, in the absence of throat-diphtheria, has led many to believe that the two diseases had different causes. Primary diphtheria of the larynx is said sometimes to extend to the throat; hence the distinction drawn between ascending and descending diphtheritic croup.

Telluric and climatic influences often have a distinct action upon primary diphtheria of the larynx. Thus, its frequency increases in proportion to the distance from the poles. It is much commoner in winter than in summer. Great dampness of the air, and prevalence of east and north-east winds, favor its development. Low, marshy spots are considered its breeding-places. It is frequent in coast-lands, sea-ports, and river-flats. It is very common on the coasts of Scotland, England, France, and Holland, the Baltic sea-coast, in many towns on the Swiss

lakes, as Geneva, and in certain parts of Switzerland and Savoy. Crawford states that it used to rage fearfully in certain marshy regions of Scotland, but became much rarer after drainage was introduced.

The primary and secondary forms are distinctly children's diseases, and attack most frequently those from the second to the seventh year. Before the end of the first year it is rare, though Bouchut has described a case in a child a week old. Adults are only exceptionally attacked. The reason for this exemption is unknown; perhaps the epithelium cells of a child's larynx are less resistant.

Boys are attacked oftener than girls; according to Rühle, in the ratio of 3 : 2.

The constitution has some influence. It is not true that the healthiest children are oftenest attacked; but scrofulous and ricketty children—or children whose parents were old, or phthisical, or marastic—are often attacked.

Hereditary influences are said to have been observed in many families, several children of one family being attacked within a rather long period of time, but this hereditary influence may be interpreted as a predisposition to this class of diseases. The same is true of the statement that children with moist eruptions of the skin often suffer with croup when the exanthem is cured.

II. Anatomical Changes.—They are almost always equivalent to those of croup in the anatomical sense; consisting of a formation of coagulated fibrinous exudation on the surface of the mucous membrane.

It very rarely happens that such a formation does not occur if the symptoms of croup exist during life; it is assumed in such a case that the mass is expectorated shortly before death.

The consistency of the exudation varies from that of thick, creamy pus to that of a hard, firm membrane; the latter may be four millimetres thick.

At first there is usually a layer like hoar-frost, or lumps of casein, in spots over the mucous membrane; the spots coalesce, and form larger ones, which may line the interior of the whole larynx, and extend to the trachea and bronchi. The membranous character is wanting, and the consistency of thick pus prevails only in the finer bronchi.

The process usually begins on the posterior lower surface of the epiglottis, extends laterally over the inner surface of the ary-epiglottic folds and arytenoid cartilage, and thence enters the larynx. If the exudation is diffusely distributed, it is usually thickest on the posterior wall of the larynx.

The exudation is usually yellowish or yellowish-gray; rarely dirty-gray, or greenish-gray, or brownish-black. It can almost always be easily removed without loss of substance, the under side often showing points and streaks of blood. The layers that lie next to the mucous membrane have the least consistency. The removal is most difficult where permanent epithelium exists, that is, on the true vocal cords. The membranes are quite loose in the air-tubes, whence they can often be withdrawn with the pincette in the form of long tubes.

Chemical examination shows that the false membranes consist of an albuminous substance resembling the fibrin of the blood. They swell in concentrated acetic acid or fluid ammonia, dissolve in solutions of alkalies or saltpetre, lime-water, or lactic acid, but are insoluble in mineral acids and cold or boiling water.

The microscope exhibits a basement substance, partly amorphous, partly

fibrous, in which round cells are distributed. The cells lie in nests or rows, in such a way as to form a sort of alternation of cell-containing and cell-less layers. A few red corpuscles are seen.

On the mucous membrane, the epithelium is gone, and more or less recognizable parts of it cling to the false membrane. Wagner first described a peculiar swelling and change of the epithelial cells, which he called croupous metamorphosis. He inferred that the membranes originated chiefly from this metamorphosis, and were, so to speak, an epithelial product. This view has been much contested, and most authors properly assume that the fibrinous mass of exudation comes from blood-vessels, the fibrin being at first fluid and afterwards coagulating on the surface of the mucous membrane.

Weigert's late researches show that the epithelial cells of the mucous membrane have some influence upon the process of coagulation, since that cannot take place unless the epithelium first perishes and enters the condition called coagulation-necrosis. A coagulated exudation, therefore, is not formed unless chemical or thermic influences (non-diphtheritic croup) or schizomycetes (diphtheritic croup) involve the protecting epithelial coat and cause coagulation-necrosis.

In describing the anatomical processes, we have anticipated the actual course of events, for croupous changes in the larynx do not begin as such, but always have an initial catarrhal stage. Redness, swelling, and increased secretion, and sometimes bleeding, are the chief signs, and they continue even after false membrane begins to form. (In the dead body they often disappear.) These conditions tend to raise and throw off the false membranes from their bed by the formation of fresh layers underneath; if the latter coagulate, the result may be a stratified false membrane.

If favorable changes occur, the deposits are sometimes expectorated as such, sometimes undergo gradual liquefaction, which facilitates expectoration. A catarrhal after-stage remains, ending in recovery.

Loss of substance and cicatrization of the mucous coat are very rare.

Changes may be found in many other organs after death. The throat is the chief seat of diphtheritic affections. Croupous processes occur in other organs—as the œsophagus, and even the stomach. The lungs are hardly ever unchanged. Collapse, pneumonic changes, emphysema (especially of the borders), interstitial pneumonia, sub-pleural ecchymoses, and œdema are variously combined. The lymphatic system is almost always involved; swelling and hyperæmia of the submaxillary, cervical, tracheal and bronchial glands, and in the glands of the mesentery, swelling of the intestinal follicles, and enlargement of the spleen are very often observed.

The corpse often presents appearances found in suffocated persons. The right heart and all the veins are full of blood, and there is venous hyperæmia in almost all the organs. There are also very frequently small extravasations in many of the viscera, and in the serous cavities there may be accumulations of bloody transudations.

III. Symptoms.—The characteristic symptoms seldom appear suddenly, but are usually preceded by warnings for some days. The child is fretful, will not play or eat; there is a little fever, sometimes repeated shivering; signs of conjunctivitis, or cold in the head or larynx, appear, the latter accompanied by cough, tickling, and hoarseness. If throat diphtheria precedes that of the larynx, the child complains of pain in swallowing or moving his head, the latter owing to painful swelling of the submaxillary lymphatic glands; or at any rate, redness, swelling, or false membrane in the throat. The more extensive a throat trouble is, the greater the danger of secondary affection of the larynx; if there are deposits on the uvula, palate, and back of the throat, the case is serious.

The special characteristic of the disease is the narrowing of the laryngeal passage, which does not become manifest until the glottis is implicated; the enemy may lurk concealed until the latter occurs. The first symptoms of such contraction, in the primary affection, usually appear about evening or midnight; in the latter case the child often wakes with the cry that he is choking. The breath (especially the inspiration) is very difficult. Inspiration is very slow, and accompanied by a peculiar whining, sawing, or whistling sound, called the stridor of croup. It can often be heard at a great distance, and has some pathognomonic value, though it occurs in other forms of stenosis. Expiration is usually free, short, and impulsive.

The dyspnœa is indicated in the position of the body, and implication of the auxiliary inspiratory muscles.

The child cannot usually lie on his back, but sits up. He often desires to change his position; now to be raised on the arm of the nurse; now to be put back in bed; or he grasps a fixed object with his arms, or plucks at the tongue and neck to remove the obstacle. Just before each inspiration the nostrils dilate. The head is bent back at each inspiration, the tongue thrust out of the open mouth, and the larynx descends deeply, rising again during expiration. The neck muscles and extensors of the back contract, to aid the efforts of the chest, and the large chest-muscles make vigorous efforts.

The degree of dyspnœa may be nearly estimated by the way the soft parts of the chest sink in during inspiration. This is due to the expansion of the thorax not being followed by that of the lungs. We see the superior clavicular fossa and the jugular fossa sink in at each inspiration. The intercostal spaces sink, especially the lower ones. In like manner the lower costal cartilages and the lowest part of the breast-bone are drawn in deeply, so that the ensiform process may come within a few centimetres of the back-bone. This is due to traction exercised by the attachments of the diaphragm, which cannot contract downwards, but is compelled to follow the suction of the chest. A deep furrow may form around the lower part of the chest, corresponding to the attachment of the diaphragm, and most developed at the ensiform process and in the lateral regions of the thorax.

The number of respirations is commonly lessened, owing to the retarded inspiration.

The voice is rough and hoarse, and if effort is made, becomes falsetto. If the disease progresses, it loses resonance and sinks to a whisper, hardly audible when the ear is held to the patient's mouth. Being unable to utter a loud sound, the patient often uses gestures, and when not understood breaks into crying.

Cough occurs at times, hoarse and barking, often called "croupy."

The pulse is usually much accelerated. Fever is almost always present, usually remittent, and without distinct type.

The symptoms may be much less severe next morning; this is not to be taken as a favorable sign, for they soon reappear with fresh severity, and cause death, either at once or after several remissions.

Laryngoscopy has been successfully performed several times. The results are tolerably uniform; there are great redness and swelling of the entire inside of the larynx, and a white coat on the mucous membrane. The symptoms of suffocation are explained by great swelling of the vocal cords, causing contraction of the glottis and respiratory immobility. From the latter circumstance we must infer that the muscles of

the vocal cords are paretic, owing to the inflammation and serous infiltration of the larynx; in fact, the laryngeal muscles are usually found very pale, moist, and swollen. If the posterior crico-arytenoids (openers of the glottis) are paralyzed, it may happen that the free edges of the vocal cords may be drawn together by suction during a hasty inspiration, so as to close the opening like a valve and threaten suffocation. This is especially dangerous in the case of children, as the opening of a child's glottis is particularly small. A croupy child, in order to avoid complete closure of the glottis, must draw the inspiration carefully and slowly; but in spite of the precaution, the contraction can only be lessened and not removed.

The state of permanent stenosis is sometimes interrupted by attacks of imminent danger of suffocation, usually due to mechanical causes. Mucous, or fibrinous membranes, detached from the lower air-passages and entering the narrowed glottis, may increase the difficulty of breathing excessively; and not in regard to inspiration alone, but also to expiration. If the foreign bodies can be removed by coughing, the respiration becomes easier. If they cannot be removed, death may occur during the attack. In other cases, the narrowing is caused by acute fibrinous exudation formed near the free edge of the vocal cords; or membranes descend from above and partly cover the glottis.

Spasm of the glottis-muscles has been thought to be a cause of these sudden attacks, but paresis is the condition of which the laryngoscope informs us. Rudnicky considers them as due to disturbed co-ordination of the breathing movements.

In croup we must never omit to examine the other organs closely.

In the throat we often find redness, swelling, and gray-white deposits, which make swallowing painful. The lymphatic glands under the lower jaw are usually swollen and sensitive to pressure.

The respiratory murmur in the lungs is usually weakened, or concealed by the laryngeal sounds. The area of cardiac dulness is often lessened, and the upper edge of the hepatic dulness lowered, showing acute inflation of the edges of the lungs. Atelectasis may be recognized by circumscribed dulness, which disappears as soon as the patient has breathed deeply, coughed, or changed position. Permanent dulness, broncho-vesicular murmur, and tinkling (consonant) râles point to a complicating pneumonia. If emphysema of the skin of the neck appears, this is referable to interstitial emphysema of the lung, extending to the cellular tissue of the mediastinum and neck, due to violent efforts at breathing.

Emphysema of the skin sometimes spreads from the wound after tracheotomy.

The barking cough usually ejects little sputum; and as children have a habit of swallowing the sputa, it is hardly ever seen.

The excretion of urine may almost entirely cease. In an epidemic which I observed at Jena, in which I made close study of the urine and its chief components, I found that not a drop of urine was sometimes produced during a whole day. The quantity of urea, sodium chloride, and phosphates is always very small. If the obstruction in the trachea is suddenly removed by tracheotomy, the quantity of urine and the

above components increases. Albumin is often present, and casts, hyaline or dotted with fat-granules, are found in the sediment.

Croup has a great influence on the temper. Children may be ordinarily obedient and quiet, but as soon as they fall ill, they become capricious, cross, and disobedient. The examination requires great skill and patience, and the administration of medicine often becomes very difficult.

The features, as a rule, are distorted, and plainly express a death-struggle.

The expression is usually livid. Great cyanosis and dilatation of the neck-veins appear when inspiration and expiration are interfered with, and the venous flow to the heart is checked.

The disease usually runs a rapid course, the result being decided between the third and the fifth day. It seldom exceeds a week, though Cadet de Gassicourt mentions a case in which croup membranes were expectorated during sixty-one days.

Recovery is exceptional. Among the sequelæ, long-contiued hoarseness and (very rarely) laryngeal stenosis have been observed.

Death occurs by suffocation in the majority of cases; it may come suddenly in an attack of "croup," or gradually, as the larynx gradually contracts. In the latter case, the skin usually becomes lead-gray or ashen-gray, consciousness is impaired, the signs of suffocation become less violent as the child is less sensible of the need of breath, and breathes superficially; vomiting often occurs spontaneously, though emetics may previously have failed to act, twitching occurs in some of the limbs, or general convulsions, and life is extinguished with symptoms of carbonic-acid narcosis. Suffocation is assisted by the existence of extensive secondary changes in the bronchi and lungs.

IV. Diagnosis.—The laryngoscope presents the means of making a certain diagnosis, but is not likely to be generally resorted to, under the circumstances. We must rely on symptoms of the stenosis of the larynx, rapidly developed. The acute, non-croupous varieties of stenosis of the larynx are chiefly acute catarrh of the larynx with great swelling (pseudo-croup), foreign bodies, œdema of the glottis, and retro-pharyngeal abscess.

Compare Vol. I., pp. 180, 182, for the differential diagnosis of pseudo-croup. The history commonly decides in regard to foreign bodies; also in œdema of the glottis, which is extremely rare in children. In retro-pharyngeal abscess, the soft fluctuating prominence on the back of the pharynx can usually be seen and felt.

Stenosis of the larynx occurring in the course of throat-diphtheria is probably due to the same cause. The throat must always be examined. False membranes may be coughed up or vomited. In doubtful cases, preserve the vomit, add water to it, and you often find fibrinous membranes floating about.

V. Prognosis.—This is very serious. Epidemics are known (Andral describes one) in which not a single child recovered. The prognosis depends on the following special points:

a. The younger the child the less favorable is the prospect, owing to the narrowness of the larynx and the feeble power of resistance.

b. Sporadic cases are usually more promising than epidemic ones.

c. It is said that croup is usually less severe in summer than in winter.

d. The presence of extensive alterations in the lungs is unfavorable.

e. The prognosis, finally, depends on the correct and decisive action of the physician, for any neglected point may cause irreparable injury.

VI. TREATMENT.—Prophylaxis is of much value. A reasonable system of hardening the body may prevent a tendency to colds or sore-throat. At places where diphtheria prevails epidemically, prophylactic gargling with salicylate of soda is advisable. Bed-chambers and living-rooms must be kept constantly aired, and in many cases carbolic spray (two per cent) may be thrown in once or twice a day. If diphtheria of the throat or larynx breaks out in the family, the affected children must be strictly isolated and, if possible, placed in another house; isolation cannot be considered complete unless the sick and the well members are under different systems of housekeeping.

The treatment has two chief objects to fulfil—to relieve existing stenotic symptoms, and to conquer the inflammation—and the two run together. For removing the croupous membranes we may use emetics or solvents.

Emetics will fail when carbonic-acid narcosis is strongly marked; yet, as Steiner observes, the admixture of wine sometimes enables them to act; or some teaspoonfuls of brandy may be taken previously. As emetics, we may use sulphate of copper (1 per cent), a dessertspoonful every ten minutes until it acts; ipecac, gr. viiss., with tartar emetic, gr. $\frac{1}{6}$, divided into three parts, one every ten minutes till it acts; or if the child resists, hydrochlorate of apomorphia by subcutaneous injection (gr. iss.: ʒ iiss., $\frac{1}{4}$ to $\frac{1}{2}$ syringe). Emetics have a merely mechanical effect, by removing the membranes like foreign bodies from the air passages. A "revulsive and corrigent" action upon the croupous process was formerly attributed to them, and the treatment of croup with emetics has been practised when no mechanical indication existed. As blocking of the glottis may occur at any time, an emetic should be kept on hand, and a trained nurse directed to use it when danger of suffocation appears.

Little can be expected of solvent remedies. Lime-water deserves most confidence; it is inhaled every hour or two from Siegle's apparatus. Biermer warmed it to increase its effect. Inhalation of lactic acid or alkaline solutions are of still less use. Muriatic acid and bromine vapors have been used in the same way.

To relieve inflammation, put the patient in a room, the temperature of which is kept steadily at 14° R. (63½° F.). The air of the room is kept moist by saucers of water set on the stove, or a two-per-cent solution of carbolic acid or lime-water is sprayed about the room ever hour or two. Give abundance of wine, milk, thin egg soup, and wine soup.

The success of internal antiphlogistic treatment is small; we simply name the chief remedies: *a.* (Locally) Ice compresses around the neck and leeches to the region of the larynx, or better, the manubrium sterni, to avoid severe bleeding from cervical veins; also, derivative friction, sinapisms, blisters. *b.* General blood-letting. *c.* Friction with mercurial ointment; calomel and sublimate internally. *d.* Hydropathic packing. *e.* Enemata of vinegar water. *f.* Pencilling the mucous membrane of the larynx with concentrated solution of lunar caustic (1 : 8) (Bretonneau), etc.

If dyspnœa increases in spite of our efforts, tracheotomy is to be employed. Do not delay too long; if extensive alterations have occurred in the trachea, bronchi, and lungs, the operation can do no good. The results would be much better, and popular dread of the harmless operation would be much less, if the right moment were not so often passed by.

We cannot wonder at failure when a child is already half dead before the operation; but the public have a tendency to lay the blame on the operating surgeon instead of the physician in charge. In spite of all this, large groups of statistics give a very fair result.

Trousseau found in	222	operations	57.2%	of recoveries.
Sanné "	4,663	"	24.0%	"
Duchek "	1,678	"	25.0%	"
Monti "	2,608	"	25.0%	"
Bartels and Wilms found in	330	"	31.3%	"
Krönlein & von Langenbeck found in	504	"	29.0%	"

Certain symptoms may require special treatment. High fever may require antipyretics; the best is antipyrin (gr. xxx. in enema). Bartels used cold baths successfully. In the narcosis of asphyxia, excitants are of use; in many cases, a warm bath with cold douche will make the patient draw deep breaths and remove obstacles to the passage of air.

3. *Nasal Diphtheria.*

I. Etiology.—This affection may be primary or secondary. The primary form is rare, and may extend down to the throat and larynx. Secondary diphtheria of the nose most frequently occurs in connection with that of the throat; and is favored by chronic inflammation of the Schneiderian membrane, and by scrofula or rickets. It is not very rare in new-born children as a result of puerperal infection.

II.—Symptoms, Diagnosis, Anatomical Changes.—The mucous membrane is greatly swollen, which, with exudation, very soon blocks up the nostrils. The patient snuffles, snores a good deal, and has to breathe with the mouth open. New-born children may suffer distress in breathing and become cyanotic, and the act of suckling may be interfered with, as they are unable to breathe except through the nose.

The interior of the nostrils may be seen to be much reddened and swollen; sometimes there are hemorrhages, but the principal thing to notice are the yellowish or grayish-green deposits on the mucous membrane, or in its tissue, associated with necrosis of the tissue. The deposits are most abundant at the choanæ.

The discharge is abundant, and may contain mucous, muco-purulent, bloody, or brownish ichorous masses, the latter often having a sickish or disgusting smell. The skin of the upper lip and parts adjoining is often excoriated, and the places swell or are covered with diphtheritic membrane. Sometimes almost the entire skin of the nose is swollen and reddened.

During sneezing and blowing the nose, deposits are often thrown out. There may be deep destruction of the mucous membrane, bone, and cartilage, and obstinate bleeding.

The submaxillary lymphatic glands are usually enlarged and sensitive to pressure.

The condition is full of danger. Death often occurs during collapse.

III. Treatment.—Irrigate the nasal mucous membrane with the nasal douche four to six times a day, with carbolic acid (gr. xxx.–℥ i. : ℥ iij.), sublimate (gr. iss.: ℥ iij.), or lime-water; in small children, syringe out the nose and use weaker solutions. If there is a discharge, insert a cotton plug and oil the nose and upper lip with borated or car-

bolized vaseline (5 per cent). A general stimulant treatment is very important.

The list of remedies given on page 380 is applicable here, with special adaptations.

4. *Diphtheria of the Œsophagus.*

I. ETIOLOGY.—The primary form is very rare; Wunderlich described it, and Steffen reported a case in which the disease spread to the throat and larynx. Extension to the stomach has even been mentioned (Andral, Steffen).

As a rule, the cases are secondary. Wagner, who has studied it closely, found it in typhoid fever, pyæmia, cholera, dysentery, measles, scarlatina, small-pox, and pneumonia. Diphtheria rarely spreads from the throat or larynx to the œsophagus. Cancer, tuberculosis, Bright's disease, suppuration in the joints and urinary passages are sometimes accompanied by secondary diphtheria of the œsophagus.

III. SYMPTOMS AND DIAGNOSIS.—Symptoms are absent, or are easily overlooked, owing to the severity of the disease. In some cases, there are very severe pains, difficulty of swallowing, or inability to swallow. Neureutter and Salmon observed fatal bleeding in a child, caused by separation of a diphtheritic deposit from an ulcer.

The diagnosis is established when the patients throw up fibrinous membranes. In Wunderlich's case, the child passed a tube-like body which presented a complete cast of the œsophagus. We should distinguish scraps of epithelium, or masses of sprue, easily recognized by the microscope.

III. ANATOMICAL CHANGES.—They comprise fibrinous deposits, sometimes within, sometimes on the mucous membrane of the œsophagus; in the latter case they are croupous; often both exist. They usually occur in spots, and are more rarely spread over the whole length of the œsophagus.

IV. PROGNOSIS AND TREATMENT.—Prognosis always serious. Treatment chiefly directed to the general affection.

5. *Diphtheria of the Stomach.*

I. ETIOLOGY.—This disease has more anatomical than clinical interest, for it is usually latent during life, and only accidentally discovered at autopsies. It is probably always a secondary affection, most frequently following diphtheria of the throat or larynx.

Many epidemics of diphtheria are remarkable for the frequent occurrence of this form, and for the fact that its extent and severity are not proportional to the process in the throat or larynx. Diphtheritic changes in the stomach occur in the course of scarlatina, small-pox, typhoid fever, cholera, dysentery, and pyæmia. It occurs in pyæmic new-born infants, whose mothers had symptoms of pyæmia before the labor, or who have developed symptoms of pyæmia or septicæmia from the cardia to the anus (Widerhofer).

II. ANATOMICAL CHANGES.—The affection almost always forms patches and islands, chiefly located in the fundus and the main cavity. These are sometimes loosely connected with the mucous membrane (croupous), sometimes can be detached from it (diphtheritic in the anatomical sense). They seldom cover a large tract, or the entire surface, of the mucous membrane. They sometimes attack the intestinal mucous membrane; in pyæmic new-born infants especially, the entire digestive tract has been found covered with a continuous croup membrane from the cardia to the anus (Widerhofer).

The consistency and thickness of the deposit vary; it may be a centimetre thick. It is yellowish, or gray, or hemorrhagic, or brownish. The microscope displays nearly parallel fibrillated masses of fibrin, containing red corpuscles, pus-corpuscles, epithelium and gland cells, and sometimes micrococci.

The mucous coat is usually swollen, hyperæmic, and studded with pus corpuscles and hemorrhages. The glands are enlarged at their bases, and the fibrinous masses extend like roots into their near extremities.

III.—SYMPTOMS. DIAGNOSIS, PROGNOSIS, TREATMENT.—The characteristic symptoms are said to be vomiting, unquenchable thirst, swelling, and pain in the stomach, but the ambiguity of these symptoms excuses us from discussing them. If croup membranes are vomited, the diagnosis may be made, if we can exclude

the larynx, throat, and œsophagus as sources. Prognosis absolutely unfavorable. Treatment symptomatic.

6. *Diphtheria of the Intestines.*

We repeat all that was said of diphtheria of the stomach. When dysentery is termed a diphtheria of the mucous membrane of the colon, we must remember that the expression is used in its anatomical, not its etiological sense.

7. *Diphtheria of the Gall-ducts and Gall-bladder.*

All the points which bear upon diphtheria of the stomach apply here. There are no symptoms, or icterus is produced by stenosis of the gall-passages, the causes of which are unknown during life.

8. *Diphtheria of the Urinary Passages.*

(*Diphtheritic Pyelitis et Cystitis.*)

Nothing in addition to what has been said within the three preceding topics.

C. ZOONOSES.

INFECTIOUS DISEASES, COMMUNICATED FROM ANIMALS TO MEN.

1. *Trichinosis.*

(*Trichiniasis.*)

I. Etiology.—Any one who eats flesh containing trichinæ which have not been killed in the process of cooking, is in danger of infection by trichinæ. The muscle trichinæ are usually inclosed in capsules; they enter the stomach, the capsules are dissolved by the gastric juice, and the trichinæ are set free. They grow rapidly in the upper part of the intestine, and develop into intestinal trichinæ. Copulation then occurs, and soon thousands of living trichinæ are born. The parents die, and are expelled with the fæces, but the young animals pass through the intestinal wall, and make their way into the muscles, where they settle, as their parents formerly did, become muscle-trichinæ, and are encapsulated. Trichina disease includes the whole period from the importation of the maternal parasite to the settlement of the young ones in the voluntary muscles.

Infection usually occurs through trichinous pork, and is commonest in regions where the use of raw pork is customary, as in the Harz and Saxony. The frequency of the disease is also affected by variations in trichinosis among swine.

In Switzerland, France, and England, trichinæ are very rare among men and animals. In Germany, Westphalia is remarkable for their rarity: the first epidemic there was observed in 1876 by Müller. In Württemberg, Häberlein described the first epidemic in 1879.

Attention has recently been called to the fact that the infection might be aided by the use of American hams and bacon, although Virchow says that this has not occurred, and that the same sometimes occurs in places which are considered free from trichina, as regards the domestic swine. The American way of fatting swine

seems to favor the spread of trichina among them, it being the practice to give the offal from the slaughter house to swine.

Eulenberg states that in 1877 2,057,272 swine were slaughtered in Russia, of which 701 were found trichinous, exactly 0.04 per cent. Billings reports examining 2,701 swine in Boston, in 1880, of which 154 were trichinous, or 5.7 per cent.

This coincides with the percentage (4.0) found in American hams and sides of bacon (Eulenberg).

If restrictions, or a veto, are laid on the importation of such wares, the Americans need find no fault.

The origin of trichinæ in the pig has been much discussed. Some think he is the headquarters of trichinæ, and that uninfected beasts acquire them by eating the droppings of the infected. It is found that all the pigs in one pen are often infected together. Infection by eating flesh of slaughtered beasts is rarer in this country.

Others believe that the rat is the original harborer of trichinæ. It is known that rats infest the pens, and that swine eat them; and trichinæ occur very frequently in rats. Zenker, however, thinks the contrary, that rats become infected by eating the dung of swine. Rogner, at Hof, found trichinæ in one pig, and also in all the rats that were caught in the same house; while the rats in neighbors' houses were free. Most recent authors, nevertheless, regard the rat as the source of trichinæ in swine, but do not wholly deny the occurrence of infection between swine.

Some other small wild animals harbor trichinæ, as the mouse, fox, hamster, badger, etc.; cats obtain them from mice and rats.

The experimental infection with trichinous flesh is successful in the case of other animals, as the ape, calf, dog, rabbit.

Goujon infected salamanders.

The larvæ of flies, eating trichinæ, digest them, but if the larvæ are eaten before the trichinæ are killed by digestion, they produce infection. Colin says that beasts of prey that live on small rodents become infected, and so do birds and fishes that eat excrement. The infection may extend still further, and the sources may become quite numerous.

The flesh of wild boars has lately been found to infect man, and in many places inspection is required by law for this meat as well as for the domestic sort. The wild animal eats some of the small creatures which we have mentioned as sometimes trichinous.

Among men the disease occurs mostly in epidemics, affecting a house, family, barrack (Kortum), or a large district. Several epidemics have sprung from the custom of making a festive occasion of slaughtering and sausage-making. A shop has sometimes infected almost all its customers. Extensive epidemics have recently occurred in Hedersleben and Hettstädt; in the former place 334 cases occurred (Kratz). In Brunswick, in 1882, an epidemic broke out which affected 254 persons (Blasius). In Emmersleben, near Halberstadt (1883), there was an epidemic of 250 cases (Brouardel).

The mode of infection has been studied since about 1860 by Leuckart, Virchow, and Zenker. In the human muscle, Hilton first described capsulated trichinæ in 1831, but took them for cysticerci. Paget discovered a spiral worm in the capsule, which Owen termed trichina spiralis. Herbst in Göttingen produced muscle trichinæ in dogs (1851) by feeding them with trichinous flesh.

The disease was known previously; there is good reason to refer certain early epidemics of rheumatoid, typhoid and sweating symptoms to trichinosis. This was directly proved at Hamburg by the autopsy of a man who survived an epidemic in 1851, but died in 1861 (Tüngel).

II. Anatomical Alterations.—Trichina spiralis is a round nematode worm. It exists in two stages, in the intestine and muscle.

The intestinal trichina may be seen with the naked eye as a very fine yellow-white thread, slightly curved or rolled at one end. The females measure three to four millimetres, while the males only reach about 1.5 mm. They have a thinner head-end and a thicker tail-end. The number of the females is always considerably larger than that of the males. They are chiefly found in the beginning of the small intestine, and only few are found in the large intestine. In examining the contents of the intestine, we dilute them freely with water and place

Fig. 69.

Fresh immigration of trichinæ into human muscle. Section of muscle near the tendinous part. Moderate power. Heller.

a drop under the microscope—unless the fine threads are visible to the naked eye. Weak powers are used (50–100 diameters) in order to give a larger field.

If muscle-trichinæ enter the stomach, and their capsule is dissolved; if they develop and copulate, it is seven days from the eating of the flesh before the females bring forth living young. The process of parturition continues some weeks, and one mother may bring forth one thousand to thirteen hundred embryos, herself dying from the fifth to the eighth week and being rejected with the excrement.

The young brood at once begins to emigrate; it pierces the intestinal wall,

sometimes turning aside at the serous coat and going between the layers of the mesentery to the retro-peritoneal connective tissue, and thence by the cellular tissue to the voluntary muscles; at other times piercing quite through to the peritoneal cavity and thence passing to the muscles through loose connective tissue; perhaps a part is carried to the muscles in the lymphatics and blood-vessels, as Virchow found trichinæ in the mesenteric glands. They are sometimes found in the flesh of such small dimensions that it seems likely that they were carried there at once by the blood or lymph.

When the young animals reach the muscles, they begin to develop into muscle-trichinæ. They pierce the sarcolemma and take up their abode inside. Inflammatory processes in the contents of the sarcolemma accompany this step. The contents become light-colored in the vicinity of the parasite, lose the transverse marking, become partly homogeneous, partly granular, and at the same time the

Fig. 70.

Isolated muscular fibre with two trichinæ. Dilatation of the sarcolemma-sac. High power. Heller.

Fig. 71.

Encapsulated muscle-trichinæ with calcification of capsule. From Heller.

sarcolemma nuclei multiply and are heaped up. The adjoining perimysium internum is also inflamed, which is recognized by the fact that the muscles become clearer at such places, and acquire a gray-red or gray-yellow tint. Before the parasites are encapsulated, they are often found stretched out, or only partly coiled up (see Fig. 69). In isolated muscle fibres a distinct enlargement and thickening of the sarcolemma-sac is present (see Fig. 70).

The muscle-trichinæ reach their full growth (0.7–1.0 mm.) within two weeks.

As a rule, only one is found in one connective-tissue capsule, two, three, or four less frequently. The capsules are elliptic or lemon-shaped, and are composed partly of a chitin-like secretion from the animal, partly of hyperplasia of the neighboring connective tissue; the trichinæ are found rolled up spirally in them, along with a granular substance. By degrees salts of lime are precipitated in the capsule, making it opaque, so that we sometimes have to dissolve the lime salt in acid before the rolled up parasite is visible (see Fig. 71). The same process may affect the trichinæ, which then breaks into small pieces. At the poles of the

capsules, outside of them, there is often an assemblage of fat-drops in old cases. The encapsulated parasite lives a great while. Klopsch gives a case in which a woman who certainly suffered from trichinosis in 1842 was operated on for cancer of the breast in 1866, when a piece of intercostal muscle was taken out and found full of calcified trichinæ-capsules, in which the creatures had retained their life for fully twenty-four years. Vitality is recognized by the parasites moving when the microscopic preparation is warmed. Müller found the capsules calcified in two-year-old pigs. The capsules are easily seen with the naked eye, as they form yellow knots of the size of poppy seeds, sprinkled in the muscles (see Fig. 72).

The capsule is dissolved in the stomach by the gastric juice, and the parasite is set free; it grows, and in two and one-half days becomes mature, that is, becomes an intestinal trichina; it copulates, and in about five days brings forth living young.

The muscles that receive the earliest and most abundant visitations are the diaphragm, intercostals, neck, laryngeal and eye muscles. The heart is free from the parasite. In the extremities they become fewer as the distance from the trunk increases. They are abundant near the insertion of the tendons, since the latter afford a natural obstacle to their further progress.

Bodies of persons who have fallen victims to the disease are often œdematous and emaciated. The arms and legs, especially the arms, are strongly flexed.

In the heart, liver, and kidney, we find cloudy swelling. Ecchymoses under the pleura, epicardium, and peritoneum sometimes occur; and more commonly on the gastric mucous membrane. The mesenteric glands are often swollen and ecchymosed.

Fig. 72.

Encapsulated and calcified muscle-trichinæ in the flesh. Natural size. After Heller.

III. Symptoms.—In some cases, trouble in the stomach or intestine begins a few hours after eating trichinous meat. The patient is sick, perhaps vomits, is sensitive to pressure over the stomach, and has diarrhœa.

In most cases, the first days pass without much trouble, but about the middle of the first week there is depression, loss of appetite, and alternating heat and chills, till at the end of the first week the symptoms are well marked.

Precursory symptoms are often absent. They include local irritation of the gastro-intestinal tract, local alterations in the muscles invaded, and general symptoms, the latter dependent partly on the changes in the digestive tract and muscular system, partly perhaps on an injurious element secreted by the parasites in the capsules, and absorbed by the stomach (Friedreich).

Nausea, want of appetite, furred tongue, bad taste, and foul smelling breath, are almost always present. Thirst is increased, from the fever and the abundant sweating. Repeated vomiting is not rare, and diarrhœa is usual; in many cases, they are so prominent as to form a "choleriform" variety of the disease. Death from collapse may occur within a few days, though that is not very common. The epigastrium is usually quite sensitive to pressure.

The presence of trichinæ in the muscles causes severe pain, easily mistaken for rheumatism, especially at the beginning of the disease. It is spontaneous, and is increased by light pressure. The muscles swell and feel hard and elastic. Very peculiar and extremely characteristic positions are gradually assumed by the limbs, and permanent flexed contracture is developed. In cases treated by me in Frerichs' clinic, the elbows were bent at an acute angle, so that the patients were unable to help themselves. This condition improves very slowly. If the eye muscles are affected, the patient complains that it is hard to move the

eye, and there are paretic symptoms or nystagmoid movements of the eyeball. Deafness has been attributed to trichinosis of the stapedius muscle. Trichinosis of the masseters produces a trismoid condition; that of the swallowing muscles, disturbance of that function; so that nutrition is severely impaired in all ways. Trichinosis of the laryngeal muscles produces paralysis of the vocal cords, with hoarseness, want of vocal power, and aphonia. Dyspnœa often occurs, due in part to disease of the diaphragm and intercostal muscles, partly to nervous causes.

Fever is one of the chief general symptoms, and often exceeds 40° C. It is usually of remittent type, and often has a temperature curve like that of typhoid. Pulse and respiration are accelerated with the increase of temperature; retarded pulse, due to disturbed innervation, rarely occurs. Œdema of the eyelids, usually a very early symptom, is very deserving of attention. Œdema of the extremities is earliest and best developed in the lower limbs; its cause has been explained by collateral œdema due to inflammation of muscles, closure of many lymphatic ducts, thrombosis of muscular veins (Colberg); during later stages, by marasmus and marantic thrombosis of veins.

The abundant sweats are in a certain sense characteristic. Obstinate agrypnia is almost always complained of. The urine is at first scanty, saturated, and often gives a brick-dust sediment. Later, from the sixth to the seventh week, Knoll observed transitory increase of urine, without change in the quantity of the most important components. Simon and Wibel profess to have found lactic acid in the urine, which they attribute to the disturbance of the nutritive processes in the muscles. Albuminuria is rare, and probably occurs only as a consequence of high fever.

The disease may be very protracted; the cause of death is usually exhaustion.

Complications are frequent. Cutaneous hyperæsthesia and anæsthesia; pain surrounding the body like a girdle (Kortum). Hemorrhage, urticaria, pruritus, furuncles, bed-sores, and pyæmia sometimes occur. Loss of consciousness and delirium may appear early, with dry, dark lips and tongue, as in typhoid; this has been called the typhoid form of trichinosis. Repeated shivering fits may occur, without special significance. Great œdema of the conjunctiva (chemosis) may occur. The fatty cellular tissue of the orbit may seem to be œdematous, causing protrusion of the globe. Kortum mentions subconjunctival hemorrhages. Kittel described one case of mydriasis which resisted suitable remedies. Pleurisy occurs rarely; pneumonia less rarely; epistaxis sometimes. Many complain of precordial anxiety; in my own patients I have seen palpitation. Kortum and others mention ascites in an epidemic in a barrack at Cologne. There is sometimes tormenting hiccup (irritation of the diaphragm). Hemorrhage from the female genitals has occurred. Menstruation is unchanged, or premature, or arrested during the disease. Kratz described two abortions in the Hedersleben epidemic; the fetus contained no trichinæ. Incontinence of the bladder is mentioned.

As sequelæ, weakness, stiffness, and pain long remain in the muscles. The skin sometimes scales off. I once observed albuminuria lasting eighteen months, and then disappearing permanently. Veh once saw œdema of the scalp followed by profuse shedding of the hair; he also describes weakness of memory.

IV. Diagnosis.—It is usually easy to determine the disease, especially in epidemics. Prominent symptoms are gastro-enteric disurbances,

œdema of the eyelids, sweats, sleeplessness, hoarseness, muscular pains, and especially continued flexion of the extremities.

In doubtful cases, we should inquire if salt or smoked pork has been eaten raw, and examine the article; or excise a bit of muscle from the patient for examination. We prefer excision to the use of the harpoon, but require strict antiseptic precautions. The wounds heal perfectly smoothly, as I have often found. Examination of the fæces is harder and less promising.

V. Prognosis.—It is serious, for we have no means of destroying the parasites after they enter the muscles, and many die after suffering great pain a long time. In the Hedersleben epidemic, 337 persons were attacked, of whom 101 died. The symptoms are severe in proportion to the number of the trichinæ. Persons infected from the same source are often affected in very different degrees. The amount eaten, the use of alcoholic drinks, and the occurrence of diarrhœa before or just after the meal, are to be considered. Nor are the animal's muscles all equally affected. The fertility of the trichina is also important: Kræmer showed that a person may have more trichinæ in his muscles than the pig from which he was infected. Children are said to be affected less.

VI. Treatment.—Prophylaxis is of the highest importance; it includes not only compulsory inspection of the pork, but previous cleanliness in the care of swine. They must be kept neat, so that they cannot eat the dung of their companions; and rats must be exterminated. If trichinosis appears, the rats must, if possible, be destroyed, to prevent communication to subsequent inmates of the pen. Raw offal, which may be infected, must not be given them to eat, nor must they be fattened in places where the flesh of diseased or dead animals is used.

Legal inspection is not a certain protection, for if an animal has only a few trichinæ, they may be overlooked, even when thorough and repeated tests are used. Yet Eulenberg's figures show how much evil is averted by the inspection. The small bit of muscle to be examined is mashed between two object-glasses and placed under a power of about fifty diameters.

Individuals should protect themselves by never eating any pork that has not been legally examined; nor ever eating raw flesh. The meat must be thoroughly cooked, especially in the interior. Vallin shows that encapsulated trichinæ are harder to kill by heat than free ones. The latter commonly die at 54 to 56° C., while the former resist to 60°. He found that three kilos of beef did not attain the temperature of 50° internally until after an hour's boiling; and required two hours more to reach 90° to 100°. Meat at 48° to 51° is often eaten; in well-done roast beef he found a temperature of 58°; in rare-done, of 51°. Twelve pounds of smoked and dried ham reached in three and one-half hours 65°, in five hours 76°, in six hours 82°, and in six and three-fourths hours 86° in the interior. Krabbe and Fjord furnish similar data.

Sausage, ham, and salt meat may also be dangerous. The process of treating flesh with wood vinegar does not kill the trichinæ. Krabbe found trichinæ still living after lying two weeks in a five-and-one-half per cent salt-brine. Colin and Fourment have done the same; the latter kept trichinæ alive a year in salt-solution.

If a person has eaten trichinous flesh, he must take a quick and powerful cathartic, *e. g.*, compound infusion of senna; a teaspoonful hourly until from four to eight full discharges have occurred, and then give a

tablespoonful of glycerin hourly till he has used about fifteen. Fiedler observed that trichinæ quickly die in contact with glycerin, even if diluted with two or three parts of water. The quicker the treatment the better.

If trichinosis is developed, nothing but symptomatic treatment remains. Friedreich recommends ℞ Kalium picro-nitricum, gr. xxx.; pulv. tub. jalap., ʒ i.; ext. glycyrrh., q. s. ut f. pil. 30. Five pills three times daily; Mosler advises benzin (f. ʒ i.–iiss. : 1 pint for enema) neither of which can destroy the trichinæ. We think it most rational to try to destroy the creatures and their offspring and expel them by ℞ Santonin, gr. ½; calomel, tub. jalap., sacch. alb., āā gr. viij. M. f. pulv. vi. S. One twice a day; but the value of this depends on not waiting too long before applying *it*—at most four to eight weeks.

Let the diet be strengthening. Give daily lukewarm baths, morning and evening, lasting thirty minutes, at 28° R. (95°). For sweating, atropia (gr. $\frac{1}{120}$ in a pill, morning and evening); for muscular pains, subcutaneous injections of morphia; and for sleeplessness, chloral hydrate (ↄ ij.–iv. per dose). Kortum uses salicylic acid, and advises not to use narcotics. Traube preferred friction with mercurial ointment.

2. *Anthrax. Malignant Pustule.*

I. Etiology.—This is the first of the infectious diseases that was proved to be caused by schizomycetes. It is most frequent in neat cattle, sheep, horses, and swine, next in deer, rarer in asses and goats. It can be transferred artificially or by accident to other animals, even to birds, fishes, and frogs (Oemler, Gibier). Graminivorous animals are more subject to it than omnivora, and the latter more than carnivora. In many places, it is endemic and destroys many beasts every year; in other places, epidemics occur occasionally.

Fig. 73.

Anthrax bacilli from the blood of a guinea-pig inoculated with anthrax. Magnified 650 diameters. After Koch.

Human beings are always infected accidentally; usually by the blood, secretions, excretions, flesh, fat, urine, etc., of affected beasts coming in contact with the skin or mucous membrane; the infection can pass through (it is said) even if the surface is intact, but as a rule, a lesion of the surface is required. Those who have much to do with animals and their dejections, as shepherds, stable boys, farmers, veterinarians, butchers, etc., are most liable to the disease. It is caught by performing autopsies on the diseased animals with cut fingers; by blood touching a cut finger while dressing the meat; by working in fat or hair from the animals; hence, it is found among leather-dealers, tanners, glovers, hat workers, wool workers, etc. It has been communicated by rags in paper-mills.

The use of meat, milk, or butter from infected animals has caused the disease in some cases, but in others it has had no bad consequences; the poison may be destroyed by cooking, or by the action of the stomach; and if there are no lesions of the mucous membrane, the poison may fail, at any rate, to enter the system.

Insects that have been upon infected animals may convey infection. Blow-flies which wet their legs and proboscis with blood have been thought especially dangerous; but other insects seem to carry contagion. The symptoms are generally said to begin with stinging pain, but that does not prove that stinging insects convey the poison, for the same sensation occurs in cases where such stings cannot have occurred.

Contagion from man to man is very rare, and has been even denied, though the blood and excrement of patients have transferred the disease to animals. The greatest number of cases in man occur in regions where it is endemic among beasts, but it is sometimes introduced from a distance, *e. g.*, by American skins. One attack does not protect against another.

The nature of the poison is known; it is connected with the bacillus anthracis, first seen by Pollender (1855) and Brauell (1857), correctly interpreted by Davaine,

and recently studied thoroughly by Koch. It is a fine, motionless rod, 5–20 μ in length, 1–1.25 μ broad (1 μ = 0.001 mm.) (see Fig. 73). It is found in the blood, especially that from the finer vessels of the viscera, and in the inflammatory products of the carbuncle and the œdema.

II. Symptoms.—If infection has occurred, the first morbid change may occur within a few hours, but in other cases the period of incubation may last seven days.

The chief visible symptoms are the carbuncle or œdema of the skin. At the autopsy, we often find disease of the mucous membrane of the intestine, which is similar to that of carbuncle. Intestinal symptoms sometimes predominate, or may exist alone. The intestinal disease has been described as mycosis intestinalis.

Malignant pustule is most frequently observed on exposed parts of the skin, to which infection has readiest access. The symptoms usually begin with stinging pain, and many patients think they have been stung by an insect. The place is reddened and infiltrated, and soon forms a distinct papule. In the middle a vesicle rises, usually not tense, filled with serum or a hemorrhagic fluid. It bursts, and the papule becomes a nodule or knob, on the border of which a ring of fresh vesicles appears. In the middle of the nodule there is a bluish-black discoloration and gangrenous destruction. The vicinity is œdematous and erysipelatous. Hard red cords (inflamed lymphatics) sometimes run from the spot, and the neighboring lymphatic glands are often swollen and painful.

By the end of the second or third day, general symptoms appear; high fever, rapid pulse, thirst, dry tongue, often hebetude, diarrhœa (sometimes with bloody discharges), swelling of spleen and liver, cyanosis, dyspnœa, and death by collapse. Tetanus sometimes closes the case. If the cause has been recognized in season, and the carbuncle rendered harmless, this general infection does not occur, and the patient recovers.

Œdema sometimes makes its first appearance at the eyes, and then in other parts. There is often erysipelatous redness of the skin, with vesicular elevations in places; blood is effused under the skin; and gangrene occurs, and in other respects the course much resembles that of carbuncle, with which this œdema is often associated.

The intestinal affection is associated with violent bloody diarrhœa, colic, pain in the abdomen when pressed, nausea, vomiting, chills, fever, increasing cyanosis and dyspnœa, death by collapse. In most cases, there is also carbuncle or œdema.

III. Diagnosis.—This is not always easy, and depends on the demonstration of anthrax bacilli in the contents of the pustule, in that of the œdema, and in the blood, but their absence is not certain evidence against anthrax. We also seek evidence of contact with portions of diseased animals. Finally, rabbits or guinea-pigs, when inoculated with the products of the inflammation, die quickly.

V. Prognosis.—There is more hope in the cutaneous affection than in that of the intestine. In the former, success depends on an early and energetic treatment by excision and cauterization, before general symptoms appear; the prognosis is serious enough at any rate.

VI. Treatment.—First and foremost, prevention of disease among animals must be attended to; for which books of veterinary surgery and public hygiene should be consulted. Great care must be exercised by those who have to do with living or dead animals who have had the disease, and in the handling of skins, hair, etc. The flesh and milk had better not be used for food, though they can be made harmless by great heat.

The treatment of the carbuncle and the œdema belong to surgery. For the intestinal complaint Leube recommended quinia and carbolic acid internally; we should prefer calomel (gr. iij., twice a day) and injections of common salt (two per cent, twice a day).

3. *Glanders.*

(*Maliasmus.*)

I. Etiology.—Glanders occurs most frequently in horses, less commonly in other monodactyla (asses and mules). It is communicable to other animals (except neat cattle), as sheep, goats, rabbits, guinea-pigs, mice, cats, dogs, etc. In menageries I have repeatedly seen lions attacked with glanders after eating the flesh of glandered horses: and the same in the case of the elephant.

Löffler and Schütz have lately shown that the cause resides in bacilli, which they discovered in the inflammatory products of the disease, cultivated them artificially, and then transferred them successfully to well animals. Morphological, though not biological, observations of low organisms in the blood and the purulent discharges of glanders were made at an early period.

Man is liable to infection, though not very prominently so. Cases occur most frequently among stable-boys, coachmen, veterinary surgeons, knackers, farmers, cavalry soldiers, horse-butchers, etc. Women are naturally less often attacked, and children still less so, except in cases where it is transmitted within a family.

The most common mode of infection seems to be by the contact of blood or secretions from diseased horses with the skin or mucous membrane. It is also supposed that the sweat, saliva, tears, and urine may contain the contagious material, but only in case the specific disease has attacked the mucous membranes concerned, or when infectious matter is mingled with their secretions. In the care of sick beasts, in cleaning and medical examination, and at the autopsy and disposal of remains, many opportunities for infection exist.

Cases are known in which persons have been bitten, and thus infected by the saliva of diseased horses.

Eating the flesh of infected animals may cause the disease, especially when it is not cooked enough.

Communication may take place between physicians and nurses in attendance on glandered men.

It is said that infection occurs by means of the air, so that it is dangerous to be in the same room with infected beasts. It is also said that no injury of skin or mucous membrane is necessary.

II. Symptoms.—The stage of incubation usually lasts from three to five days, but is said sometimes to extend to weeks.

The symptoms include nodes or diffuse infiltration, consisting of round cells, occurring on the skin and in the muscles, in the mucous membrane of the nose, frontal sinuses, throat, larynx, trachea and bronchi, and in the viscera, as the lungs, liver, spleen, kidneys, stomach (Wyss), and even in the central nervous system. Both nodes and infiltrations have a great tendency to caseous changes, suppuration, calcification, and a very slight tendency to cicatrization; if one spot begins to cicatrize, another continues to form new knots and infiltration, with destruction of tissue. Ulcers of the skin are thus formed, which continuously extend, so that the cutaneous disease has been popularly called the "worm."

There is an acute, a subacute, and a chronic form of glanders. While acute glanders usually terminates within a week or two, the chronic form lasts as many months or years, or even longer than ten years. Acute symptoms sometimes appear, changing a chronic into an acute disease; the converse probably does not occur.

Acute glanders often begins gradually. If the poison is received through a wound of the skin, a lump or ulcer appears, which soon becomes discolored, has a disposition to spread rather than heal, and discharges thin hemorrhagic pus, of an offensive smell. In the neighborhood there is often inflammatory œdema or erysipelas, and inflammation of the lymphatics and neighboring lymphatic glands. Similar changes soon appear in other parts of the skin. Vesicles appear in places, or impetigo and ecthyma, which form the starting-point of new ulcers.

Chills and febrile movement sometimes occur, with a feeling of weakness, and pain in the muscles and joints. There may be swelling or suppurative inflammation of joints. The appearance of the patient, with sordes on the lips and tongue, delirious and unconscious, may sometimes remind one of typhoid fever.

If the mucous membrane of the nose is also attacked, the patient complains of burning, dryness, and pain in the forehead; thin, purulent bloody masses are discharged, sometimes with a foul smell. The septum narium may be destroyed.

Ulceration and inflammation may extend to the mucous membrane of the mouth and throat; the submaxillary lymphatic glands swell, and sometimes form abscesses. Disease of the laryngeal mucous membrane and lungs are known by pain, difficulty in swallowing, hoarseness, cough with fetid expectoration, and perhaps œdema of the glottis. There is usually loss of appetite and constipation; in the later stages, diarrhœa.

The liver and spleen are sensitive to pressure, and often enlarged. In the urine, albumin often occurs; leucin and tyrosin have been found (Ninaus). Death usually occurs from exhaustion.

In chronic glanders, the symptoms are nearly the same, but take a slower course, with frequent remissions and exacerbations.

III. Anatomical Changes.—We have mentioned them briefly. Numerous abscesses are often found in the body, occupying the skin, muscles, mucous membranes, lungs, liver, and spleen. Hemorrhage in any organ may occur. The nodes vary from the size of a lentil to that of a man's fist, and much larger. Bone and cartilage are often involved in the breaking down of the lumps.

IV. Diagnosis.—There is often a difficulty in distinguishing glanders from pyæmia, and in chronic cases from syphilis or tuberculosis.

A possible opportunity for infection is to be most carefully inquired into. The diagnosis may, perhaps, be made more easily and safely by means of Loffler's and Schütz's bacilli.

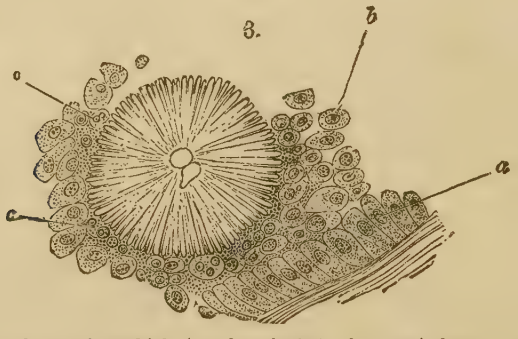
V. Prognosis, in acute glanders, almost always unfavorable; in the chronic cases, there are fifty per cent of cures.

VI. Treatment.—Chiefly confined to the case of external disease; incision of abscesses, carbolic dressing, and carbolic injections for the nose and throat. Food light, but strengthening; alcoholic drinks. Iodide of potash, strychnia, mercurials, etc., are not likely to be of use. For the rest, purely symptomatic treatment.

4. *Actinomycosis.*

Originally confined to the bovine race, it occasionally attacks man. It is caused by a radiating fungus, actinomyces (see Fig. 74), which forms firm grains,

Fig. 74.



Actinomyces grain from a bronchiole (cut lengthwise) of a cow's lung; *a*, epithelium of the bronchial mucous membrane; *b*, epithelioid cells; *c*, round cells. In the middle, the radiating fungus. Magnified about 350 diameters. From Marchand.

that can be crushed, and gives to the diseased products a peculiar character. The fungus is probably one of the moulds. Actinomycosis is probably the same in man and in cattle; the fungus has been successfully inoculated from the former to the latter. In man, most cases produce multiple abscesses and pyæmia, but internal organs, as the lungs, bronchi, intestine, pleuræ, peritoneum, and others, may be invaded. Israel has lately compiled thirty-eight observations, and has endeavored to form a clinical outline of the disease. A few cases seem to be different from the rest; but the majority begin at the mouth, air-passages, and intestine. Actinomycosis of the bronchial mucous membrane may cause putrid bronchitis; that of the lungs constitutes chronic infiltration of the lung tissue, leading to cavities, destruction, especially pleurisy and metastatic abscesses in other organs. Peritonitis and metastatic disease often accompanies actinomycosis of the intestine. Death is the usual result; the disease can be recognized during life only by the grains, which are easily distinguished by the naked eye. Treatment purely symptomatic.

5. *Hoof-and-Mouth Disease.*

Aphthæ Epizooticæ.

I. Etiology.—The ox, sheep, and pig are most frequently attacked: it is rarer in the goat, horse, or the dog, or fowls. An acute febrile disease, leading to the

formation of vesicles on the mucous membrane of the mouth, also between the toes, and on the teats; it is communicable by the contents of the vesicles, and by the urine, dung, blood, and milk.

Several cases are reported in the human race, usually after eating raw or insufficiently cooked milk, or in milkers who touched the udders of diseased animals, covered with vesicles, with sore hands. Chance may cause infection in other ways, *e. g.*, by the slaver falling on one. Eating butter or cheese may communicate the disease.

II. Symptoms.—The period of incubation lasts three or four days; then fever occurs, and yellow vesicles form on the mucous membrane of the lips and tongue, more rarely on the hard and soft palate, which burst in a few days and leave erosions of the mucous membrane. There is a burning and heat in the mouth; sometimes great swelling and trouble in swallowing. At nearly the same time vesicles appear between the fingers and toes, and around the nails. A vesicular eruption has been seen on the breasts; in a few cases, a general exanthem. Signs of gastro-enteritis sometimes occur, which, in connection with fever, may destroy small children. In the middle of the second week, the vesicles dry up, form thin crusts, and fall off without scars. The disease usually ends in two or three weeks.

III. Diagnosis, Prognosis, Treatment.—In diagnosis, the history is of special importance. Prognosis is good; death is rare. Milk from diseased animals must not be used at all unless it is thoroughly boiled; cuts must not be soiled with secretions or excretions from diseased animals. For treatment, wash or pencil the mouth with chlorate of potash (1 : 30); or lime-water, a teaspoonful every hour if diarrhœa is present. Vesicles on the extremities are to be rubbed with fat and covered with salicylated cotton.

6. *Rabies, Hydrophobia.*

I. Etiology.—This disease is chiefly confined to dogs, but also occurs in cats, horses, oxen, sheep, asses and mules, wolves, foxes, hyænas, badgers, martens, and jackals. It does not occur spontaneously; it is almost always spread by the bite of mad animals. Saliva and blood contain the infectious material, while flesh and milk have repeatedly been eaten without injury.

Hydrophobia in man is usually caused by the bite of dogs; less often by that of cats, foxes, oxen, etc., or by autopsies of mad animals, or by accidental contact of saliva or blood with wounds. Not all persons that are bitten become hydrophobic; the infecting saliva may be intercepted by the clothes. Some lesion (perhaps a very trifling one) appears to be always necessary to receive the poison.

A spontaneous occurrence of the disease has been affirmed, even for man, until very recently, but all the evidence is untrustworthy. Communication from man to man is not certainly known.

Klebs suspects that the poison resided in brown schizomycetes, which he demonstrated in the salivary glands of Professor Hermann, who died of hydrophobia in Prague.

II. Symptoms.—The period of incubation varies very much, but usually lasts from fifteen to thirty days, and oftener longer. It is said that the period has been protracted, not merely to one or two years, but from ten to thirty years, but the latter belongs to the realm of fable. A period of six months is established with certainty.

There is usually a short stage of premonitory symptoms before the outbreak of the disease, lasting from one to three days, on an average. The bite, if not healed, begins to give pain, to swell, bleed, and discharge more freely, or the cicatrix, if formed, becomes sensitive, livid, and is said to open sometimes (?). Pain often radiates from the wound over the whole extremity to the spine. There is deep depression of mind; the patient grows pale, restless, loses appetite and sleep, and is usually tortured by unspeakable anxiety about the disease. The pupils are enlarged, the gaze is fierce, and symptoms of the spasmodic stage soon appear.

Spasms of inspiration may be among the first of these symptoms. These are deep, sighing, or sobbing inspirations, during which the patient becomes cyanotic and is greatly distressed for breath, and feels suffocated. Spasms in the act of swallowing occur when the patient tries to eat or drink. The sight, or even the thought of drink, or the mention of it, may produce attacks of such spasms. The patient is unable to swallow his saliva, which is secreted in excess, and has to

eject it from time to time. The tendency to spasms of breathing and swallowing increases; at last a mere draught of air, jostling of the bed, mental excitement, or bright lights produce it. The temperature rises to 39°, 40°, and higher. The pulse increases in frequency, up to 100–120. Albumin and sugar have been found in the urine. Constipation is common.

Psychical changes gradually develop; delirium, hallucination, and distinct attacks of rage. The patient is maniacal, striking, spitting, reviling those about him; if the attendants use force, spasms occur in some extremities, or general convulsions, and many make snapping movements like those of biting. Such attacks may recur at short intervals. In their free intervals the patient is conscious, and begs pardon for his violence.

Death may occur suddenly in such attacks, which vary between one-half and three-quarters of an hour. Or they cease, giving place to symptoms of collapse and death, which usually comes within two to four days.

III. Anatomical Changes.—There is hardly anything characteristic. Rigor mortis is usually well marked. Decomposition usually begins very soon. The blood is remarkably thin and dark. Lütkemüller has found the white blood-corpuscles increased, and numerous microcytes.

In the membranes of the brain and spinal cord, there is usually œdema, and often hemorrhage; the latter also in the substance of brain and cord, and the neurilemma of peripheral nerves. The follicular apparatus of the back of the tongue and the fauces has been described as swollen. The mucous membrane of the stomach and intestine has ecchymoses in places. The spleen has been noticed as swollen. In the liver and kidneys, parenchymatous cloudiness and fatty changes have been seen. The peripheral lymphatic glands have been occasionally found enlarged and ecchymosed.

IV. Diagnosis.—The disease is easily recognized, especially when the history is clear. Though many authors in recent times have denied the existence of the disease, and have declared that it is merely tetanus, there exists this distinction. that in tetanus there are no complete intermissions; nor are there mental disturbances.

V. Prognosis.—Bad; very few cases of cure are claimed.

VI. Treatment.—Let prophylaxis be the first step. High taxes should lessen the number of dogs kept; a muzzle should be worn by every dog, so constructed as completely to prevent his biting; when hydrophobia appears, let all dogs be shut up strictly for at least six months.

If a man is bitten by a mad dog, let him at once suck the wound, and have it cauterized as soon as possible by the hot iron or caustic potash to destroy the poison.

If the disease appears, give narcotics energetically. Among the best means is continued chloroform-narcosis, during which the spasms cease. The injection of morphia, clysters of chloral and curare are less certain. The other narcotics are probably too weak. No specifics are known, however loudly some may be praised. The calm assurance of the physician is above all important.

Pasteur reports that inoculation is a protective to the human species.

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